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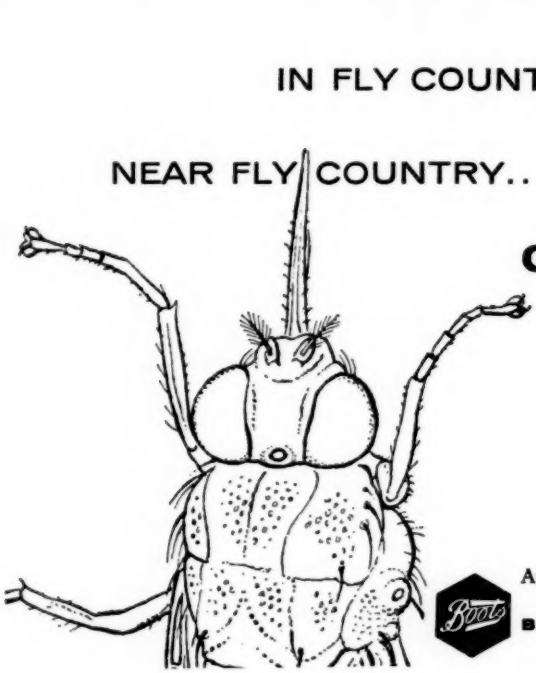

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Congress Papers

SYMPOSIUM ON DISEASES OF SHEEP*

(1) The Prevention of Pulpy Kidney Disease

BY

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SUMMARY.—Pulpy kidney disease cannot be regarded as an infectious or contagious disease in the ordinarily accepted use of these terms. It is a toxaemia arising from the absorption of the toxin of Clostridium welchii *type D, produced in the gut when the intestinal contents are of a nature conducive to rapid growth of the organism.*

A large and high quality food intake appears to be the main factor which creates the favourable intestinal conditions, but this is the basis of modern intensive methods of sheep management. Very often the management conditions accepted as essential to economic production of lamb and mutton are just those which set the stage for a high incidence of pulpy kidney disease.

Methods of management which may be of value in the prevention of this disease are essentially those which check thriving and, therefore, defeat the objective of the flockmaster. In these circumstances it is suggested that the rôle of the veterinary surgeon is to meet the risk created by the desirable environment by establishing and maintaining a high level of immunity in all the sheep all the time.

Specific antiserum is recognised as the method of establishing a short-term immediate protection in the face of losses from pulpy kidney disease, but it is pleaded that continuous protection must be the objective, and suggestions are made for the use of vaccine to this end.

IN making a contribution to this Symposium on Diseases of Sheep I have been asked to speak to the title "The Prevention of Pulpy Kidney Disease." I think it best to get some reasonably good definition of the condition before dealing with its prevention. The term pulpy kidney disease was coined in New Zealand before the nature of the condition was understood. When it was recognised as due to absorption of *Cl. welchii*, type D toxin from the intestine and known that it occurred in sheep of all ages—outside the original age group of about 8 to 12 weeks—the term entero-toxaemia was adopted. It is certainly a more appropriate name, but I imagine pulpy kidney disease will remain the popular designation of the condition for many years. Although I, personally, think it an unfortunate title, it is somewhat to be preferred to the popular name which almost crept into being through these sudden deaths in young lambs having been ascribed in Britain to wool-ball. In any case, it is best to recognise at the outset that we are dealing with a disease which, although of bacterial origin, cannot be regarded as infectious or contagious in the generally accepted meaning of these words. The organism which produces the causative toxin is a normal inhabitant of the environment of sheep and is commonly present in the intestines of normal animals. It is only when some unusual condition in the intestine encourages rapid multiplication and massive toxin production that the disease occurs. We can broadly say that abnormal conditions arise when there is stasis in the intestine or when the contents are of a nature conducive to the abundant growth of *Cl. welchii*. There is little doubt but that the intake of increased quantities of protein rich food—whether it be in the form of milk, young rapidly growing grass or the hand feeding of concentrates—is the precipitating cause. It is interesting to speculate why to-day the age of incidence of pulpy kidney disease is, apparently, so much extended beyond what it originally appeared to be. Perhaps it is the old, old story that interest in, and search for, the presence of any disease reveals its occurrence to an extent hitherto unobserved. Perhaps modern methods of pasture management and the economic necessity to keep young sheep growing and fattening to the upper limit are the more important factors; certainly it is the rapidly thriving

* Symposium of papers presented to the Annual Congress of the British Veterinary Association in Glasgow on Thursday, September 1st, 1960.

lamb which is likely to succumb to pulpy kidney disease and it is in flocks being well managed for economic production that the highest incidence is likely to occur.

It has long been accepted that losses from enterotoxaemia can be controlled by methods of management. The penning of ewes with thriving lambs at foot and the movement of flocks to less luscious, even bare, pastures are practices which have been adopted with some success. The avoidance of movement to better pastures has for long been accepted as a way of avoiding sudden deaths. However, surely the practise of these methods, if they be accepted as good management from the prevention of pulpy kidney disease point of view, are the antithesis of good management with its sights set on economic production of high quality carcases at the earliest possible age. Modern methods of pasture management and sheep husbandry are aimed at maximum economic production. They must bring with them increased risk of disease occurrence and, particularly, pulpy kidney disease. Is it not one of the primary functions of our profession to meet the risks associated with high level production by means which do not interfere with that objective? On all sides, whether in poultry, pigs, sheep or cattle we hear modern intensive methods blamed for the increase in illness and death among stock. We should, and I am sure we shall, always declare our intention to meet that challenge by understanding the risks created by intensive management, and by finding ways and means of avoiding disease losses without interfering with what appears to be an economic necessity of these days. It is against this background that I find myself unable to accept that we should meet the threat of pulpy kidney disease by suggesting methods of management which depress the rate of growth and fattening of sheep and lambs.

We can accept as reasonably certain that the conditions which produce good thriving in the lamb tend to be associated with those for good thriving of *Cl. welchii* type D in the intestine and, with its multiplication, the circumstances associated with mass absorption of toxin and the sudden death of the host. I contend, then, that it is our clear duty to provide the sheep or lamb with protection against this toxaemia—with the ability to thrive and grow fast unassociated with the risk of pulpy kidney disease. We can only do this by specific immunisation.

I have the feeling that in the majority of our intensively managed flocks the risk of pulpy kidney disease is almost constantly present, and I suggest in these circumstances that the target should be to keep the stock highly immune at all times. We may not strike our target but we should at least aim at it.

I have had the privilege over the years of watching the progress which has been made in the improvement of the implements and methods of immunisation. I am very satisfied that the high quality sera and vaccines which are available to-day provide the weapons with which we can achieve our objective. The great difficulty, as I see the position, is to determine how best to use them in all the variety of management methods which seem characteristic of British sheep husbandry. However, if we do accept that the objective is to keep all the sheep highly immune to the toxin of *C. welchii* type D all the time this problem is simplified.

With that objective in mind one sees only a minor rôle for hyperimmune antiserum. It has a place, to meet the emergency in unvaccinated sheep and to cover short periods while active immunity is being established by the use of vaccine. I suppose it is far too much to expect that the day will come when all the flocks in Britain are thoroughly immunised—when flock-masters have accepted that they must think and act months ahead rather than take action after the stable door has been pushed ajar. Certainly when it has been pushed ajar, by the loss of one or two lambs from pulpy kidney disease, the immediate use of specific antiserum can be the means of preventing its flying open by the occurrence of a quite serious level of loss among the best lambs in the flock.

Let us concentrate on how best continued active immunisation can be achieved. It is generally agreed that the injection of a single dose of even to-day's quality vaccine cannot be expected to do more than prepare the sheep for true full active protection. It is evident that some sheep which have never been vaccinated carry a low level of active immunity. It probably arises from an occasion on which moderate growth and toxin production by *Cl. welchii* type D has occurred in the intestine with the absorption of a non-lethal amount of toxin—enough to stimulate some antibody production. Such sheep will make a sufficient response to the injection of a single dose of vaccine but they must be ignored since they can only be identified by laboratory tests. Incidentally, I would interpolate my belief that the credit given to single dose vaccination has probably arisen from its use in flocks in which many of the sheep have absorbed a non-lethal dose of toxin from the intestine in a symptomless attack of the disease and developed a low grade basal immunity. But this level of immunity is near nothing in comparison with that which can be achieved by vaccination. There is clear evidence that if a second dose of vaccine is given after an interval of one month or longer, the response to that second dose is many-fold higher and in the majority of sheep to a level which will afford protection for a period of about 6 months. The sheep previously unvaccinated cannot be accepted as protected until about 10 days after it has had a second dose of vaccine—the interval between the doses being, for best response, at least one month. From that point forward the giving of a third or subsequent dose of vaccine will further, and quickly, enhance and prolong the level of immunity? Perhaps it is at this point that I should put in the " escape clause." An occasional animal will not make the normal response to the injection of antigen, hence I would not maintain that in all sheep given two properly spaced doses of vaccine the immunity so produced will be sufficient to cover for a 6-month period the most massive absorption of toxin conceivable. In any immunisation

scheme one could never guarantee 100 per cent. protection, but in this case I am satisfied one is high up in the nineties.

With the objective of converting an unvaccinated flock to an immune one, where and when should one start? I am inclined to answer, "anywhere, any time," for much will depend on the time of year at which we are approached for advice and action. However, pulpy kidney disease is such a terror to the lowland fat lamb producer who purchases cast ewes from the uplands that I illustrate my suggestion by assuming that we have a flock of ewes in the autumn, of at least doubtful history as to vaccination. Vaccinate all of them and give a second dose in the spring, preferably about 10 days before lambing, unless the ewes are to be moved to a better pasture prior to lambing when the second dose should be given before the move is made. These 2 doses should protect the ewe through the spring months and allow her to pass to the lamb *via* the colostrum sufficient antibodies to protect it for the first 8 to 12 weeks of life. That temporary immunity in the lamb should be converted to an active one by the use of vaccine, giving one dose when about 4 weeks old and a second after an interval of 4 to 6 weeks. By this means one has established an immune flock. How does one keep it highly immune? Our experience is perhaps not sufficient to permit the recommendation of only once-a-year vaccination thereafter—the giving only of the spring dose before lambing. That may be sufficient but, for the present, I would prefer that both the autumn and spring doses are repeated year by year, and since the timing is not critical these injections can be given on occasions when the sheep are gathered for other purposes—provided always that the necessary time and care are taken and a suitable location used. Obviously, sheep subsequently introduced into the flock must be immunised as were the original flock. One has great confidence that the adoption of this procedure will keep the flock free from loss from pulpy kidney disease, but should some confirmed pulpy kidney loss occur the indication is that an enhanced immunity is necessary and the injection of an additional dose of vaccine at once can be depended upon to push the immunity to a much higher level within only a few days of its injection.

If the general principles on which the above outline is based are accepted as sound, the scheme can easily be adapted to suit the circumstances in individual flocks and to meet those peculiar to the season at which one is asked to direct the establishment of an immune flock.

It is very probable that some will find my next text—that the objective should be to keep all the sheep immune all the time—unacceptable for one or more of a variety of reasons—economic, troublesome, even impracticable. In intensively managed flocks producing fat lambs as economically as possible, I would not accept any of these excuses for doing nothing until trouble, in the shape of sudden deaths, descends on the flock. Certainly they are not acceptable excuses for not proffering advice along these lines, but we all have to help the flockmaster who carries to us his tale of woe, his shout for something to be done in the face of serious pulpy kidney losses. The use of antiserum is the obvious answer in these circumstances for one cannot depend on the flock having a "natural immunity" as the result of the absorption of a non-lethal dose of toxin and so containing a really high proportion of individuals ready to make a full response to a single dose of vaccine. I believe that in these circumstances the veterinary surgeon is amply justified in trying to get his client to take the long view—in advising that a dose of vaccine be given on the occasion of the injection of serum and that it be followed up by a second dose about one month later to establish an immune flock, and that thereafter revaccination be practised to avoid a repetition of the crisis.

The theme of my paper has been so simple and the suggestion so direct that some may be disappointed that I have not covered a wider field—diagnosis and differential diagnosis (perhaps particularly the confusion which sometimes arises between pulpy kidney disease and hypomagnesaemia in adult sheep), enterotoxaemia due to *Cl. welchii* type C and, possibly, even lamb dysentery. I have even omitted to discuss the risks of unfortunate local infection associated with the use of pulpy kidney vaccine, as with any vaccine. I feel confident that you, Mr. Chairman, will use your authority and discretion to guide the discussion: to allow it to flow beyond the title of the paper if you think this wise and desirable. I have steeled myself against wandering into these associated fields and held firmly to the subject which your Provisional Committee invited me to cover.

FOOT-AND-MOUTH DISEASE

As we go to press we are advised by the Ministry of Agriculture that in the past few days, over 20 outbreaks of foot-and-mouth disease have been confirmed in England. These have included animals sold in markets in Hexham on October 27th, Richmond on November 2nd and Ponteland on October 31st and November 7th. Animals in transit through markets at Malton on October 29th and Beverley on November 2nd have also been exposed to infection. In order that all animals exposed in these markets may be inspected as soon as possible, the whole of the counties of Northumberland, Durham and the North and East Ridings of Yorkshire have been declared a Controlled Area in which the movement of animals is prohibited.

The restrictions came into force at midnight on November 8th and will remain in force until the risk of spread of infection is past.

The Controlled Area comprises the administrative counties of Northumberland, Durham, North Riding of Yorkshire and East Riding of Yorkshire, including the cities and county boroughs of Newcastle upon Tyne and Kingston upon Hull, and also the city and county and county borough of York, and the Parish of York Castle.

(2) Sudden Death in Sheep

BY

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SUMMARY.—The importance of flock history, management, season of the year and relation to parturition in the diagnosis of the cause of sudden death in sheep is discussed. The conditions commonly associated with sudden death are listed according to their seasonal occurrence and the age of animal affected. Diagnosis of the individual diseases under field conditions is considered with a description of the post-mortem *lesions and confirmatory tests.*

The relative economic importance of the diseases leading to sudden death are discussed. The incidence of the various causes as encountered by a diagnostic laboratory in south-east Scotland are tabulated.

IN the diagnostic laboratory sheep are by far the predominant species presented with the legend "found dead." The sheep is peculiarly susceptible to changes in diet and management, and the illnesses which result, both bacterial and metabolic, often have a course of only a few hours. Some 2 decades ago this syndrome presented no difficulty in diagnosis —it was braxy. To-day the position is vastly different with a much greater knowledge of the varied diseases involved. This, however, presents the problem of differential diagnosis and it is with this aspect that it is proposed to deal in this paper. The diseases considered are those which definitely can result in sudden death. Though many other cases are recorded as "sudden," this is often the result of faulty observation and such cases have been omitted.

In no species is a full and accurate history of more importance in arriving at a diagnosis. It is an unfortunate fact that it is often impossible to make a definite diagnosis on a *post-mortem* examination alone, *e.g.* hypomagnesaemia; but when the *post-mortem* findings can be related to a full history, not only of the animal in question, but of others which may have been affected, diagnosis may be attempted with a reasonable expectation of accuracy. It is in fact often possible to narrow the probabilities to one or two on history alone and use the *post-mortem* examination for final determination. The fact that a carcase has been submitted with the short history of "found dead" does no preclude the amassing of a considerable amount of relevant information from the carcase. Unfortunately a full history usually requires close questioning of the stockman as the information volunteered is rarely that required. While this is relatively simple in the field it is more difficult at the laboratory where all too often the sheep is brought in by a carrier, who is unable to help. The flock history is the first point to be considered. This is important not only from the diagnostic point of view but also to assist in the formulation of prophylactic measures. Certain diseases are seasonal and this narrows the number to be considered. The age and breed of animal affected and its bodily condition help to limit the field further. This information can be determined by the investigator when a carcase is submitted but not from organs. He is left with a number of subsidiary questions to which he should have the answers and it is here that the greatest difficulty arises as so many questions are the outcome of a negative or affirmative answer to previous questions. One difficulty too frequently encountered is the state of decomposition in the carcase. The further advanced this is the more impossible it is for a satisfactory *post-mortem* examination to be carried out. In succeeding paragraphs when *post-mortem* lesions are discussed it is assumed that the carcase is being examined within an hour or two of death.

It is helpful to consider the common causes of sudden death by listing them under season and age.

Spring

In-lamb ewes: hypocalcaemia and other metabolic upsets, Clostridial infection of the foetus, acute pasteurellosis and pneumonia, prolapse of the intestines through the vaginal wall and, rarely, pulpy kidney disease.

Lambing ewes: hypocalcaemia, hypomagnesaemia, blackquarter metritis and pasteurellosis.

Post-lambing ewes: hypomagnesaemia, hypocalcaemia, pulpy kidney disease.

Lambs: lamb dysentery, pulpy kidney disease, bacterial septicaemias.

Hoggs: pulpy kidney disease, pasteurellosis, hypocalcaemia.

Summer

Pulpy kidney disease, red gut.

Autumn

Hoggs: braxy, pasteurellosis.

Ewes and hoggs: acute fluke and black disease, pulpy kidney disease. It will be obvious from this list that at any time of the year only a limited number of conditions need be considered. When the improbables have been eliminated it is the points of differential diagnosis in those diseases left which will be considered below. The histories and findings quoted are based on experience in South-East Scotland and some of the observations will not apply under different systems of management.

The "In-lamb" Ewe

Hypocalcaemia

The most common cause of sudden death before lambing is hypocalcaemia. Symptoms and "suddenness" are, however, both complicated by hypomagnesaemia and hypophosphataemia. The most important factor in this syndrome at this stage of pregnancy is the hypocalcaemia in the low ground sheep. Hypocalcaemia and hypomagnesaemia either

together or separately appear equally common in hill ewes. If several animals, however, have been found dead and none noticed ill the indication is either that some condition other than hypocalcaemia is involved or that the intervals between the shepherds' inspections are too long. This condition—*i.e.* hypocalcaemia—does not often kill a ewe suddenly; several hours usually elapse between the onset of symptoms and death. The length of time between inspections must, therefore, be ascertained and if necessary more frequent visits advised. This will usually result in a sick animal being observed. In this disease response to treatment is a very important diagnostic aid, and response by the hypocalcaemic sheep to an injection of calcium borogluconate is sufficient to clinch a tentative diagnosis. If the opportunity is presented to the veterinary surgeon to obtain a blood sample from an affected but still living animal biochemical analysis will, of course, furnish additional confirmation. Such a sample should always be taken, as, if the animal fails to respond to treatment the diagnostician has obviously a second chance to find the real cause of the illness. An oxalated sample is also useful. The cast hill ewe carrying a half-bred lamb on arable grazing is particularly susceptible to hypocalcaemia. This is also true of hill sheep which are suddenly brought from the hill to low pasture for lambing (Hughes & Kershaw, 1958). When a carcase is examined *post mortem* the condition is readily differentiated from foetal clostridial infections and pasteurellosis. Some difficulty might, however, be encountered with pulpy kidney disease, but it is the writer's experience that this disease is rare before lambing and usually can be considered unlikely. The differential diagnosis is considered in the post-lambing group.

Pasteurellosis and Pneumonia

A certain amount of true pneumonia with grey hepatisation of part of the lung is a common finding in the cast hill ewe on low ground. It is important that undue significance is not attached to this lesion. This is particularly important where the case could be one of hypocalcaemia. The lesion may, however, be associated with an acute flare-up resulting in a septicaemia with *Pasteurella haemolytica* the organism involved. This condition can result in sudden death. The lesions usually found are an area of complete consolidation and grey hepatisation with an adjacent area of active pneumonia or perhaps intense congestion. Care must be taken to differentiate these from the passive or hypostatic congestion often seen in an animal which has been recumbent for a time—a mistake which is by no means uncommon. The general *post-mortem* picture in acute pasteurellosis where solidification of the lungs is rare, is as described for hoggs by Stamp *et al.* (1955) and discussed briefly in this paper under pasteurellosis in hoges. The diagnosis is confirmed in the laboratory by isolation of *P. haemolytica* from the organs and heart blood of the affected animal. It is noteworthy that this disease also, though bacterial in nature, is often precipitated by environmental factors, in particular when old hill ewes are grazed on a lush highly manured pasture or the hill ewes are brought down to lamb.

Other Conditions

The other two diseases mentioned—clostridial infection of the foetus, and prolapse of the intestines through the vaginal wall—are readily diagnosed and cannot be confused with any other condition. The former—named "rotton lamb disease" by the stockowner—is characterised by grossly swollen and gassy foetuses obviously infected by anaerobes. Such deaths can often be controlled by prophylaxis with blackquarter serum. The rupture of the vaginal wall with extrusion of the intestines is usually seen in the bigger ewes of the larger breeds. Death is sudden when the extrusion occurs. The cause is unknown.

The Lambing Ewe: Blackquarter Metritis

While the ewe at this time does, of course, die from the conditions mentioned above, an additional source of loss is blackquarter metritis. This disease is usually referred to by shepherds as "inflammation" and this name also covers death from metritis following the non-specific infections of retained placentas, etc. True blackquarter metritis commonly occurs immediately after parturition and death is invariably sudden. *Post-mortem* findings are oedema of the perineal tissues, the vaginal wall and the wall of the uterus. Gas is sometimes found in the lesion. While *Clostridium welchii* and *Cl. septicum* may be involved in some cases the success attendant on the use of *C. chauvoei* antiserum prophylactically indicates that this organism is the primary cause of most outbreaks. The condition can usually be halted by administration of serum to all ewes still to lamb, with a penicillin injection at all assisted parturitions. It may be necessary, however, to change the lambing field.

Post-lambing Diseases

Hypomagnesaemia and Hypocalcaemia

Sudden death in the ewe from lambing to 3 or 4 weeks after lambing presents the most difficult problem in differential diagnosis that is met in sheep practice. Generally speaking hypocalcaemia is a disease of the first few days after lambing and is rarely a really "sudden" death, symptoms usually showing for a few hours before death supervenes. Hypomagnesaemia tends to occur later from 1 to 4 weeks post lambing when milk yield is reaching its peak and the animal is found dead or *in extremis*. Again, of course, the 2 conditions can co-exist. It is noticeable, however, that hypomagnesaemia in the ewe is much more common on young highly manured grass and less frequent on older pasture. The same diagnostic criteria as have been discussed above apply to post-lambing hypocalcaemia. Response to treatment by the hypomagnesaemic ewe is very poor.

Differentiation of the 2 conditions at *post-mortem* is practically impossible, the differences in typical cases being of degree only and in the absence of a blood sample from a sick animal diagnosis must be based on a consideration of all the facts available.

The diagnostic problem is further complicated by the possibility of enterotoxaemia—a disease which

is by no means uncommon in the lambed ewe, particularly the hill ewe being "done well" on inby pasture. There are, however, some differences in the *post-mortem* findings in cases of hypomagnesaemia and enterotoxaemia, the 2 conditions most likely to be confused.

In hypomagnesaemia the small intestine can show a length of intense congestion, but usually the major portion of the bowel is milk white in colour with a tracing of red blood vessels prominent on the surface. Small subserous haemorrhages are present at the ends of the branches of the blood vessels. In pulpy kidney disease the bowel does not usually show any great change. The kidney lesion is, however, probably the most useful guide in distinguishing between the metabolic diseases and enterotoxaemia. In hypomagnesaemia the kidneys are usually very pale and firm with a varying amount of petechiation and blood splashing. This latter is occasionally so severe as to make the kidney cortex appear very congested but rarely does this extend to both kidneys. The appearance is similar in hypocalcaemia, but usually only some petechiation is seen on the cortex, rarely any extensive haemorrhages. This differs markedly from the port-wine coloured kidney of enterotoxaemia where the cortex is usually disintegrating and may be readily washed away by running a gentle stream of water on to the surface. The other lesions found are common to both conditions—myocardial haemorrhage, excess pericardial and pleural fluid, etc.

In the laboratory, examination for toxins in the intestinal contents can be helpful. Smears from the same contents will often show a preponderance of clostridial organisms present and the presence of sugar in the urine is considered to be diagnostic. If the carcases are at all decomposed *post-mortem* examination is unhelpful. The mere presence of toxins, however, should not be taken to establish a diagnosis as Bullen (1952) has shown that these toxins can be present in potent amounts in normal sheep. Toxins can often be demonstrated in hoggs passively immunised with serum where death was, in fact, due to acute pasteurellosis and in some of the lambs affected with acute nematodiriasis (Stevens, 1959). The methods of detecting toxins and their value in diagnosis have been discussed by Stevens (1959). The same author emphasises the limitations of *post-mortem* and biological tests without adequate histories, and it is considered unnecessary to expand on this in this paper.

Lambs

Apparently sudden deaths recorded in lambs up to a month old are frequently the result of faulty observation. *Post-mortem* examination will often give the clue to this in the presence of abscesses, empty stomachs, etc., showing that the lamb must have been ill for some time. These indications, especially the empty stomach, must always be looked for. The 3 conditions listed—bacterial septicaemias, lamb dysentery and pulpy kidney are considered the common causes of real "sudden death." The differentiation of the clostridial diseases from the septicaemias give little difficulty on *post-mortem* examination in the young lamb. Most difficulty is probably experienced in differentiating acute lamb dysentery and pulpy kidney disease. This can be done satisfactorily only by demonstrating protection to the specific toxins. Pulpy kidney—usually regarded as a disease of the older lamb—has not infrequently been encountered in lambs under a week old and thereafter is quite common. The stomach in these cases is usually very full. Lambs more than a week old are unlikely to die suddenly from lamb dysentery, and in any case the extensive ulceration which is readily seen at *post mortem*, and which is always present in such older lambs, leaves no doubt as to the cause of death.

Bacterial septicaemias are involved in a significant number of deaths in young lambs and often they are not observed to be ill. Laboratory examination and culture are essential to determine the cause of death. This examination also allows the sensitivity of the causal organism to antibiotics to be determined. The frequency with which *Escherichia coli* is now found is noteworthy. These conditions are usually best dealt with by changing the lambing field, or where this is impossible, prophylactic antibiotic therapy is indicated.

Summer

Apart from "exotic" diseases which are not always easy to diagnose—and sometimes are not diagnosed at all—the majority of the sudden deaths at this period are due to enterotoxaemia. After handling, the disease referred to as "red-gut" may be seen in one or two lambs in a flock and in the occasional ewe. *Post-mortem* examination shows the intestinal tract as a whole, or for a major part, a dark red colour and usually distended with gas. The condition bears a strong resemblance to that seen in pigs (Jennings, 1959). We have consistently failed to demonstrate toxins from the usually blood-stained bowel contents, nor have we been able to demonstrate potentially dangerous *Clostridia* in significant numbers from such cases. Losses are, however, invariably insignificant in numbers.

Braxy and Pasteurellosis

In autumn the lambs or hoggs—usually from hill farms—may suffer from braxy. Perhaps as a result of widespread vaccination the disease is comparatively infrequently encountered and is almost never seen in low ground flocks. Feeding hoggs being grazed on aftermath and rape also suffer from acute pasteurellosis. Acute fluke and black disease are the other 2 conditions which must be considered, ewes as well as hoggs being liable to die from these causes. Pulpy kidney disease may, of course, still be present and is usually the first condition to be considered.

The differential diagnosis does not present so much difficulty as the spring series does especially if more than one specimen can be made available.

Braxy

A diagnosis of braxy depends on the demonstration of the dark haemorrhagic lesion in the abomasal wall. Confirmation is by examination of a frozen section from the edge of the lesion. No other con-

dition gives the picture presented by this condition. While the general *post-mortem* picture is that of an "anaerobic death" the abomasal lesion can often be seen when the abdomen is opened, and the carcase has the characteristic smell associated with *Cl. septicum*. This condition when the carcase is fresh should not be confused with either black disease or pulpy kidney; but as mentioned earlier, decomposition can render exact diagnosis impossible.

Pasteurellosis presents a *post-mortem* picture of an acute bacterial disease with haemorrhages and blood-splashing in the connective tissues and on the serous surfaces. The lungs do not collapse, and when removed the marks of the ribs can often be plainly seen. The surface is slatey blue in colour, mottled, and with marked petechiation. The cut surface exudes blood and froth in quantity. Confirmation is by the isolation of *P. haemolytica* from the organs. The animals concerned are usually being kept under conditions liable to result in pulpy kidney disease and toxins can frequently be demonstrated from the intestinal contents. It may then be difficult to determine the exact rôle played by these two entities as the presence of the toxin may be adventitious. As, however, the only really effective prophylactic measure to control pasteurellosis of this type is to administer a severe check to the animals by immediately moving them to a bare pasture or confining them for part of the day, the question is of more academic than practical importance as this treatment will also control enterotoxaemia. Black disease and acute fluke can be difficult to differentiate, and in fact diagnosis of black disease depends on the demonstration of the characteristic lesion in the liver with confirmation by means of a frozen section. The presence of immature fluke is, of course, common to both conditions. The liver damage and reaction to their migrations may mask the black disease lesion. The demonstration of *Cl. oedematiens* toxin in the peritoneal fluid may help in differentiation. The constant liver damage simplifies the differentiation from pulpy kidney disease, but the mere fact that numerous immature fluke are present does not rule out the possibility of enterotoxaemia being responsible for outbreaks, and this point should be considered and an examination for toxins of *Cl. welchii* type D carried out.

Lastly, in severe weather in winter, sudden deaths found to be associated with hypocalcaemia may occur in hoggs. This condition is by no means rare. Pulpy kidney disease may cause losses in feeding hoggs when concentrates are stepped up early in the year.

Conclusion

This review covers the important diseases causing sudden death in sheep. Other causes do, of course, exist, and reference to them will be found in the literature. Such conditions are either rare and sporadic or strictly local in their occurrence, *e.g.* struck, and are well known to workers in the affected areas. They have, therefore, been omitted from this paper.

The economic importance of the conditions mentioned is very difficult to assess. Epidemics appear and disappear suddenly on individual farms and while one of the less common diseases may cause heavy losses to the individual farmer their overall significance is not high. The most striking disease is, of course, pulpy kidney disease or enterotoxaemia which is undoubtedly a disease encouraged by modern methods of intensive farming. This disease occurs at most times of the year in one class of sheep or another and the overall losses must be heavy. The predisposing cause to sudden death in the sheep in most of the conditions listed is undoubtedly management. It is necessary, therefore, for the veterinary surgeon dealing with sheep problems to have a sound knowledge of the husbandry methods practised in his area. Such knowledge is essential not only for diagnosis but for formulation of advice on control measures.

In the list below are tabulated the diagnoses made at the Edinburgh Veterinary Investigation Centre in the past 2 years in disease outbreaks when the syndrome was characterised by sudden death. It should be noted that these figures are no indication of the numbers involved in each outbreak, ranging as they do from single cases in the miscellaneous and red gut categories to as many as a score in the hypomagnesaemia outbreaks.

Pulpy kidney disease or enterotoxaemia	102
Lamb dysentery	8
Braxy	6
Black disease	4
Blackquarter metritis	4
Redgut	9
Hypocalcaemia	25
Hypomagnesaemia	35
Hypocalcaemia and Hypomagnesaemia	15
Acute fluke	5
Acute pasteurellosis	38
Lamb septicaemias	15
Miscellaneous	20
Clostridial infections of foetus	8
Prolapse of intestines	4

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HOUGHTON POULTRY RESEARCH STATION

We are advised that with effect from December 1st, 1960, the Station's charge for a *post-mortem* examination will be increased to 15/-. This charge will cover the examination of one bird or, in certain circumstances, will cover a group of related birds or chicks sent in at the same time from any one outbreak.

As a disease precaution the Station cannot accept any *live* specimens for clinical observation.

(3) Experimental Scrapie: Some Recent Work

BY

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SUMMARY.—Recent experiments using sheep-passaged and goat-passaged sources of scrapie are described.

Using Cheviot sheep as test animals the effect of boiling the inoculum has been further investigated. This material has been shown to be active when administered subcutaneously. Dilution and serial transmission experiments are described. No cases of scrapie occurred in animals injected with autoclaved material.

An attempt to demonstrate activity in brains of normal sheep is described.

The addition of adjuvants to scrapie sheep spleen and nodes prior to injection did not appear to influence the process.

The above findings are discussed.

Introduction

CUILLE and Chelle in a series of papers (1936, 1938a, b, 1939) claimed to have transmitted scrapie to sheep and goats. Since that time most of the published work on biological transmission has been carried out at either Moredun Institute, Edinburgh, or the A.R.C. Field Station, Compton, and the results have been published in a series of papers. The most recent review is that of Palmer (1959). Since that time 4 further publications have appeared in which experimental transmissions are reported (Stamp, Brotherston, Zlotnik, Mackay & Smith, 1959; Pattison, Gordon & Millson, 1959; Darcel & Avery, 1960; Pattison & Millson, 1960). The purpose of the present contribution is to record some recent observations on the experimental disease.

Materials and Methods

These were essentially those used by Stamp *et al.* (1959).

Sheep. The animals used were of the Cheviot breed and except where stated were between 6 and 12 months old when inoculated.

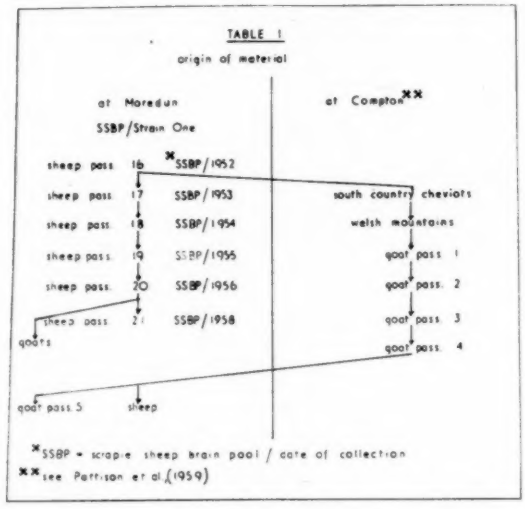
Source of material. The normal sheep brain pool used was the same as that used by Stamp *et al.* (1959) and is referred to as NSBP/1955.

The source of scrapie brain pools is shown in Table 1. The "strain" used was that isolated by Wilson *et al.* (1950) and is referred to as SSBP/Strain 1.

Inocula. All brain pools were prepared by homogenising without diluent in an M.S.E. "Atomix" at 13,000 r.p.m. for 2 minutes. They were then distributed into wax cartons and stored at −35° C. A 10 per cent. w/v suspension of homogenate in an 0.85 per cent. solution of sodium chloride in distilled water was prepared immediately prior to use. This was centrifuged at 500 g. for 30 minutes. The supernatant fluid was collected and this is referred to as the 10^{-1} dilution and constituted the inoculum unless otherwise stated.

TABLE 1

origin of material



Boiled material. The 10^{-1} dilution was then placed in a flask which was completely immersed in a beaker of water (see Fig. 1). The temperature of the water in the beaker was raised to boiling point (the temperature of the contents of the flask = 99 to 99.5° C.). Boiling with constant stirring, using a magnetic stirrer, was continued for 30 minutes. Unless otherwise stated the boiled product was then centrifuged at approximately 500 g. for 10 minutes and the supernatant fluid was collected. This material constituted the inoculum and is referred to as the 10^{-1} dilution (boiled).

Experiments with Sheep and Goats

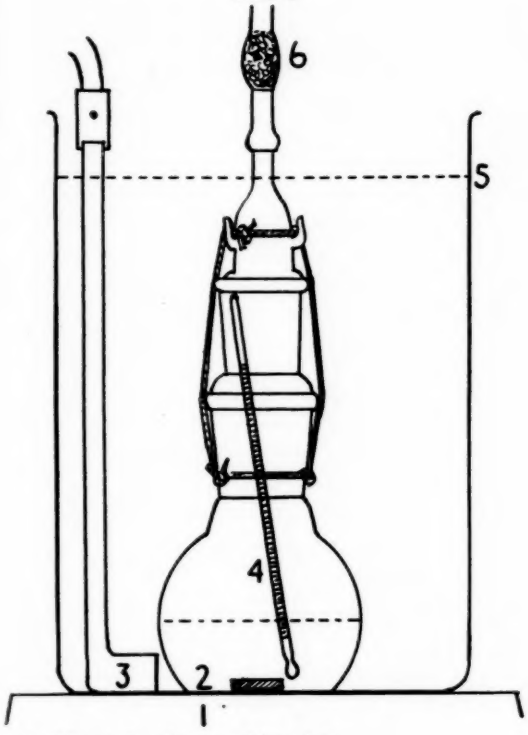
One of the most interesting results of recent years has been the demonstration of the apparent high degree of susceptibility of the goat to the experimental disease (Pattison *et al.*, 1959). This finding, coupled with the fact that the disease has only once been recorded as occurring naturally in that species (Chelle, 1942) would appear to indicate the goat as the animal of choice for experimental work. It is, therefore, of interest to examine the differences, if any, between sheep and goats as experimental subjects.

Scrapie Sheep Brain in Goats

In May, 1957, 5 goats were each inoculated intracerebrally with 1.0 ml. of a 10^{-1} dilution of SSBP/Strain 1/Pass. 20. Two cases of scrapie developed

in this group at 29 and 32 months respectively and a third case is suspected at 35 months (April, 1960). Of 15 day-old lambs inoculated with the same material 6 cases of scrapie developed within 5 to 6 months after inoculation.

FIG. 1



1. Magnetic stirrer and hotplate.
2. Stainless steel magnet.
3. Immersion heater.
4. Thermometer.
5. Water level.
6. Air filter.

A group of 6 goats and 15 day-old lambs were each inoculated intracerebrally with 1.0 ml. of a 10^{-1} dilution (boiled) of SSBP/Strain 1/Pass. 20 at the same time as the above group. At 35 months (April, 1960), no cases of scrapie have been confirmed in these goats while 2 cases of the disease have been diagnosed at 9 and 11 months in the lambs.

In March, 1958, a further group of 6 goats and 9 sheep were each inoculated intracerebrally with 1.0 ml. of a 10^{-1} dilution (boiled) of SSBP/Strain 1/Pass. 20. Scrapie was diagnosed in two sheep at 9 and 12 months, but there have been no cases in the goats to date.

Scrapie Goat Brain in Sheep

Two goat brains (C4 and C45) from a fourth passage of scrapie in goats at A.R.C. Field Station, Compton, were obtained (see Table I). These were pooled and a 10^{-1} dilution was used as inoculum.

Thirty sheep and 3 goats each received 1.0 ml. of this inoculum intracerebrally. The 3 goats were diagnosed as cases of scrapie 8 months after inoculation. Four sheep were thought to be showing early signs of scrapie at 7 months but the disease did not appear to progress. At 12 months, however, 2 of these 4 sheep have now been confirmed clinically as cases of scrapie.

Experiments with Sheep

One of the main stumbling blocks to the acceptance of a viral aetiology of scrapie is the apparent resistance of the agent to various treatments but in particular to boiling. The following experiments were undertaken to confirm and possibly elucidate earlier work on this characteristic of the agent.

Subcutaneous Inoculation of Sheep with Boiled Scrapie Sheep Brain

A 10^{-1} dilution (boiled) of SSBP/Strain 1/Pass. 20 was prepared. A dose of 5.0 ml. of this material was administered subcutaneously to each of 10 sheep. As a control 1.0 ml. of the same preparation was given intracerebrally to each of 10 sheep.

Three of the sheep which received the subcutaneous inoculation developed scrapie within 7 to 9 months, while 2 which received the intracerebral inoculation developed the disease in 6 and 7 months respectively.

Dilution of Boiled Scrapie Sheep Brain

A 10^{-1} dilution (boiled) of SSBP/Strain 1/Pass. 21 was diluted with an 0.85 per cent. solution of sodium chloride in distilled water so as to give dilutions of 10^{-2} and 10^{-3}. The dose of inoculum per sheep was 1.0 ml. and this was given intracerebrally. Thirty sheep were inoculated at each dilution.

In the 10^{-1} group 6 cases of scrapie developed between 4 and 9 months following inoculation, while in the 10^{-2} group there was 1 case at 9 months and in the 10^{-3} group 1 case at 7 months.

Serial Transmission of Boiled Scrapie Sheep Brain

A serial passage of boiled scrapie sheep brain was initiated in 1957. Ten sheep were each inoculated intracerebrally with 1.0 ml. of 10^{-1} dilution (boiled) of SSBP/Strain 1/Pass. 20. There were 2 cases of scrapie in this group 6 and 7 months after inoculation. The brains of these 2 cases were pooled and a 10^{-1} dilution (boiled) was prepared in the usual way. Each of 30 sheep was inoculated intracerebrally with 1.0 ml. of this material.

Eight cases of scrapie developed in this group within 5 to 8 months following inoculation.

Effect of Autoclaving on the Scrapie Agent

A filtrate of scrapie sheep brain was autoclaved

PAPERS FOR THE HYPOMAGNESAEMIA CONFERENCE

All the papers available in advance to those delegates who have asked for them were posted yesterday (Friday). If any do not arrive early in the week will members kindly write to the Editor of THE VETERINARY RECORD.

at 20 lb. p.s.i. (126.5° C.) for 30 minutes. No cases of scrapie occurred in 17 sheep inoculated intracerebrally with this material (Wilson, 1954).

To confirm this finding a further experiment was carried out in which a 10^{-1} dilution of SSBP/Strain 1/Pass. 21 was used. This material was autoclaved at 20 lb. p.s.i. The coagulum which formed was resuspended in the fluid phase by grinding in a Griffith tube and the whole material constituted the inoculum, 1.0 ml. of which was given intracerebrally to each of 30 sheep. No cases of scrapie developed within 12 months following inoculation.

It should be noted that the 10^{-1} dilution (boiled) used in the "dilution of boiled scrapie sheep brain" experiment described above was prepared from the same starting material.

The Addition of Boiled Scrapie Sheep Brain to Normal Sheep Brain

From the above results there is thus ample confirmation of earlier work demonstrating the resistance of the agent to boiling. One hypothesis is that the action of boiled material is to stimulate a "latent" virus already present in a proportion of apparently normal sheep. The following experiment was undertaken in an attempt to demonstrate the presence of such a virus in a pool of 20 apparently normal sheep brains (NSBP/1955).

Preparation of boiled material for use in this experiment is essentially that given under "Materials and Methods" except that the coagulum after boiling was resuspended in the fluid phase by grinding in a Griffith tube.

Group A. Equal parts of boiled SSBP/Strain 1/Pass. 20 prepared in this way and a 10^{-1} dilution (unboiled) of NSBP/1955 were mixed prior to injection. The inoculum consisted of 1.0 ml. of this mixture given intracerebrally within 12 hours of birth, to each of 50 lambs.

Group B. Equal parts of the same boiled SSBP/Strain 1/Pass. 20 suspension as was used above and boiled NSBP/1955 prepared in the same manner were mixed prior to injection. Fifty lambs were each given 1.0 ml. of this mixture as in Group A.

TABLE III

Inoculum	No. of animals	No. of cases	Incubation in months
boiled scrapie brain + unboiled normal brain	50	7	7-9
boiled scrapie brain + boiled normal brain	50	6	6-10
unboiled scrapie brain only	20	3	5-7
boiled scrapie brain only	9	2	9 & 12
unboiled normal brain only	20	0	—

Group C. The potency of the untreated SSBP/Strain 1/Pass. 20 was similarly tested in 20 new-born lambs.

Group D. The potency of the boiled SSBP/Strain 1/Pass. 20 prepared as above was checked in a group of 9 sheep.

Group E. The inability of the unboiled NSBP/1955 to produce scrapie has already been reported by Stamp *et al.* (1959).

The results of this group of experiments are given in Table II, from which it can be seen that there is no significant difference between groups A and B.

The Effect of Addition of Adjuvants

The pooled scrapie spleens and nodes used in this case had already been shown to be infective (Stamp *et al.*, 1959). The normal spleens and nodes were obtained from apparently normal abattoir sheep. Fifty per cent. w/v tissue suspensions in saline were prepared by homogenisation in the atomix. The resultant mixtures were not centrifuged but mixed with Freund-type adjuvant in the proportion of 1 part tissue suspension to 5 parts of adjuvant. All injections were intramuscular and each sheep received a total of 6.0 ml. distributed over 4 doses at weekly intervals.

Group A consisted of 15 sheep given scrapie tissue + adjuvant.

Group B consisted of 15 sheep given normal tissue + adjuvant.

Group C consisted of 15 sheep given physiological saline + adjuvant.

Two cases of scrapie have occurred in Group A at 6 and 9 months following the first injection. No cases of the disease have developed within 9 months in the other 2 groups.

Discussion

The results of the present work with scrapie in goats are of interest in that they suggest that a change may have taken place in the behaviour of the agent after its passage through goats. Sheep material gave rise to scrapie in 6 of 15 lambs within 6 months following inoculation, and 2 of 6 goats at 29 and 32 months respectively. After 4 passages from goat to goat the picture appears to have reversed and 3 of 3 goats injected were actually fully developed cases at 8 months, while even at 12 months after inoculation, only 2 cases were clinically confirmed in 30 sheep given this material. It is also relevant that whereas passage of brain material from goat to goat has resulted in a decreased incubation period (Pattison *et al.*, 1959), there was no significant difference in incubation between a first isolation from natural cases (Wilson *et al.*, 1950) and the 19th serial passage of this strain from sheep to sheep (Stamp *et al.*, 1959). It is particularly necessary that later goat passages should be checked in sheep since if adaptation were indeed taking place this might have important implications.

Pattison and Millson (1960) have shown that the optimum conditions for producing scrapie in goats are provided by intracerebral injection of a suspension of scrapie goat brain and that, if the preparation of the inoculum or the route of inoculation is varied.

there may be a significant lengthening of the incubation period and perhaps a reduction in take. It is, therefore, worth pointing out that in the present experiments with sheep and those reported earlier (Stamp *et al.*, 1959), cases of scrapie occurred within 12 months whichever route of parenteral injection was used and despite various treatments, the only exceptions being those in which normal material or autoclaved scrapie sheep brain was used as the source of the inoculum.

It is of course realised that by imposing an arbitrary time limit of 12 months on our Cheviot sheep experiments some cases of induced scrapie will escape detection but, on the other hand, when sheep of 6 to 12 months of age are used our results are unlikely to be confused by naturally-occurring scrapie in our experimental stock since the natural disease rarely occurs in animals less than 2 years of age. If our experimental animals were observed for longer periods it is considered that such confusion could readily arise, either because the sheep were not clear of the disease when purchased, or because of possible contact with the disease at the Institute, for such contact has been previously reported (Greig, 1940). However, the missing of a few cases of scrapie is only of importance where negative results are recorded, and since up to the present it has been particularly difficult to obtain negative evidence when using scrapie material in Cheviot sheep, our methods would appear to be justified when investigating the agent in such sheep. On the other hand if sheep other than Cheviots are used the position is not so clear for we have evidence (Stamp & Dickinson, 1960) that the inoculation of our Cheviot scrapie pool material into 6-month-old Suffolk sheep did not give rise to scrapie within a 12-month period and this was so whether the sheep inoculated were born from scrapied or non-scrapied parents. A similar long incubation period is reported by Darcel and Avery (1959) when they inoculated Suffolk sheep since they assumed that the disease which developed in their inoculated sheep was the result of their inoculum although they pointed out that this was not necessarily the case. If and when scrapie does occur later in such animals one is never sure whether the disease has developed as a result of the inoculation or is the natural disease. It would seem therefore that not only is there variation between goats and sheep in response to the inoculation of scrapie material but also between breeds of sheep.

Some of the differences between goats and Cheviot sheep as test animals have been discussed and it will be obvious that further knowledge of these differences is required before the goat can be relied upon as the sole test animal for all purposes.

One of the first steps in the investigation of a filterable pathogen is to attempt to characterise the agent. In addition to demonstrating that it gives rise to pathological effects it is usually necessary to show that it has specific morphological or serological characteristics. Attempts to demonstrate such characteristics have been singularly unsuccessful with scrapie. It seemed to us, however, that the most outstanding feature of scrapie is the apparent resistance of the agent to boiling. Before accepting that scrapie is due to a self-replicating agent which will withstand boiling it was considered essential to investigate a number of other possible explanations.

One of the most obvious possibilities is that the intracerebral inoculation of boiled material mechanally disrupts the blood brain barrier and thus allows invasion of the CNS by a factor present in other tissues of a proportion of sheep. This seems unlikely in view of the fact that neither normal nor autoclaved scrapie brain material introduced in this way induces the disease, but the results of the present work showing that boiled material is active when introduced subcutaneously makes such a hypothesis untenable.

Further, if the boiled material simply acted as a stimulating agent and did not itself replicate then its activity would probably be rapidly lost on dilution. The present experiments show it to be active at least to a dilution of 10^{-3}, the maximum dilution tested.

Before accepting that the agent which withstands boiling is self-replicating it is, of course, necessary to establish that the material remains active even when boiling is carried out at each passage interval during serial transmission. This has been shown to be the case for 2 passages to date and this experiment is being continued.

It is possible that the scrapie agent, even if heat sensitive, might be protected in some way in the brain material used as the source of inoculum. Certain streptococcus phages when present in cream with 50 per cent. fat content require steam at 100° C. to inactivate them (Whitehead, 1944). In view of the high lipoid content of brain it is necessary to show that the scrapie agent is equally resistant to boiling in other tissues than brain before accepting that it has itself these characteristics. Such experiments are in progress.

Since it had been shown that the unboiled agent was filterable and serially transmissible (Wilson *et al.*, 1950) it was considered possible that the action of the boiled agent might be to activate, or be reactivated by, a virus normally latent in the tissues of a proportion of sheep. It was further presumed that if such a latent virus did exist it would possess characteristics of heat resistance of the customary range (*i.e.* it would be destroyed by boiling for 30 minutes) and the samples of 20 normal brains used should be large enough to include one or more animals carrying the postulated virus. If this theory were correct it might have been expected that in the group of animals given boiled scrapie plus unboiled normal brain there would have been a higher proportion of takes than in the group given boiled scrapie plus boiled normal brain. It can be seen from Table II that there is no significant difference between these groups and it has not been possible with the techniques used, to demonstrate the existence of such a "latent" virus in a pool of 20 normal brains.

From the results of previous work (Stamp *et al.*, 1959) there is good reason to believe that experimental allergic encephalitis and scrapie are quite

(*Concluded at foot of page* 1006)

(4) Anaemia in Trichostrongylid Infestations

BY

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SUMMARY.—The anaemias associated with trichostrongylid infestations are reviewed and the nature of the pathogenic mechanisms discussed. The evidence is confined mainly to the species Haemonchus contortus *and* Trichostrongylus axei. *It is suggested that gastric haemorrhage and dyshaemopoiesis of alimentary origin may both play a part but, while the last is characteristic of* T. axei *infestations, the anaemia of haemonchosis is predominantly of the first type. The evidence is examined which relates infestation size and blood damage quantitatively. Close correlations are described between helminth reproductive activity and the degree of red-cell reduction in the post-haemorrhagic anaemia of haemonchosis, but the quantitative aspects of the other types of anaemia remain to be worked out. It is concluded that in haemonchosis the reproducing adults play a relatively much greater part in the genesis of anaemia than do the developing larvae.*

PARASITIC infestations of sheep are often characterised by symptoms of anaemia of one kind or another. Examples which readily come to mind, where anaemia is a regular feature, include heavy infestations with flukes, *Fasciola hepatica;* nodular worms, *Oesophagostomum columbianum;* hookworms, *Bunostomum trigonocephalum* and *Gaigeria pachyscelis;* the strongyle *Chabertia ovina* and certain of the Trichostrongylids among which the stomach worms *Haemonchus contortus* and *Mecistocirrus digitatus* are outstanding.

Of the other members of the Trichostrongylid family we have much less direct information relative to the production of anaemia. Although it appears not to have been convincingly demonstrated in all cases, no dissent has been registered from the opinion that the pinkish coloration so frequently observed in them is due to contained blood (Ross & Gordon, 1936; Lapage, 1955). Among the probable ingesters of blood there may, therefore, be included species of the genera *Ostertagia, Cooperia* and *Nematodirus.* Regarding the genus *Trichostrongylus* opinions differ: Gibson (1954) excluded the possibility of blood-sucking from his discussion of the effects of *Trichostrongylus axei* in experimental infestations, and Andrews (1939) produced no evidence of the habit in mixed experimental infestations of the intestinal species (*T. colubriformis* and *vitrinus*), on the other hand Ross and Gordon (1936) consider that "there is little doubt that they suck blood." Such inconclusive evidence clearly leaves us a long way short of assessing the relative importance of the various members of the family in the causation of anaemia in the host. Field observations and records leave little doubt that some of them play a part, and that not all the anaemias seen in association with parasitic gastritis and enteritis are due to *H. contortus.* From such findings, moreover, the weight of suspicion appears to fall in particular on the *Ostertagia* spp. and *T. axei.* Unfortunately, parasitic nematode species never

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Experimental Scrapie.—*Concluded.*

distinct entities. It still seemed possible, however, that the same underlying mechanism might be responsible, *i.e.* an immunological response possibly of the delayed hypersensitivity type. If this were the case the addition of adjuvants might be expected to influence the process. A source of scrapie other than brain was used for this purpose so as to avoid the complication of allergic encephalitis. The course of the disease was not influenced in the speed at which it developed nor was there an increase in the proportion of takes in the experiments reported above.

The experiments described in this paper demonstrate the complexity of one aspect of the problem of characterising the agent of scrapie. There is no evidence, up to the present, that the agent is other than a self-replicating substance which can withstand boiling but, as pointed out above, further work is necessary before this can be accepted without reservation.

Acknowledgments.—We wish to thank Dr. W. S. Gordon and Mr. I. H. Pattison of Compton for their co-operation in providing the 4th scrapie passage goat brains used in the experiment reported above.

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occur singly and naturally acquired infestations comprise a range of species whose numerical ratio varies from time to time, place to place, and animal to animal. This obviously limits the definitive conclusions which can be reached concerning pathogenesis under field conditions since there is no means of apportioning the rôle as agent to one or other species, or their interactions.

The alternative approach is through experimental infestation under controlled conditions, but for various reasons comparatively few such studies have been carried out. The expense and labour of raising the animals free of worms and the facilities required for their maintenance are important limiting factors, but apart from these there are other problems. Trichostrongylid species vary considerably in the facility with which they may be cultured to the infective stage and with which they may be stored in adequate numbers in readiness for experimental work. Moreover, the establishment of experimental infestations is much less certain with some than with others. Ostertagia species are notorious in the difficulties they present in this respect. The published information on the effects of experimental infestation is, therefore, not only meagre in quantity, but it relates to very few species. The present discussion is, consequently, restricted mainly to the species *H. contortus* and *T. axei*.

The questions to which we seek an answer are: first, what is the mechanism by which the observed blood changes are produced? Second, how are these changes related quantitatively to the infestation which produces them? On the first question histological studies on blood and bone-marrow have contributed very valuable presumptive findings, but too often a wealth of patiently acquired histological detail has been married to quite inadequate helminthological information, and presented without other essential information relative to such things as age, history and nutritional status of the subjects. On the second point very little evidence is available. Quantification has long been the aim of helminthologists, but the problems involved demand such large resources in facilities and personnel with the consequence that too often well-intentioned projects have terminated in results which are inseparable from the operations of chance.

From the earliest days, *e.g.* Railliet (1895), Veglia (1915) and others, the blood-sucking behaviour of *H. contortus* has been regarded as its most significant feature. Anaemia thus raised no difficulties since it appeared to be an obvious consequence of the blood-letting sustained by the host. Doubts as to the adequacy of this as a complete account of the pathogenesis began to appear, however, in relation to the parallel situation of hookworm infestation of dogs and man and in turn affected views on ruminant stomach-worm infestations. Schwartz (1921) drew attention to the anaemias symptomatic of infestation with non blood-sucking worms such as *Diphyllobothrium latum*, providing presumptive evidence of toxic factors. He identified erythrolysins in a number of taxonomically unrelated worm species including *H. contortus*. In this species the erythrolysin was so weak as to discount it as of more than incidental significance. Nevertheless, the possibility became admitted that the pathogenesis of anaemia in worm infestation might include factors other than the simple direct one of blood loss.

Fourie (1931) undertook the first serious study of anaemia in sheep experimentally infested with *H. contortus*. He used 38 animals which he dosed with various specified numbers of infective larvae, but the rates and levels of dose were apparently arbitrarily chosen and not based upon a systematic rationale. Unfortunately, since this was the only helminthological information presented, the work, in spite of a wealth of haematological detail, has only a qualitative significance. Anaemia was observed in 15 of the animals, of which 9 cases terminated fatally and 6 recovered. It was characterised by a rapidly progressive oligocythaemia which in the later stages became megacytic and hypochromic. There was no evidence of damage to the red cells, neither was erythrophagocytosis observed, nor did any of the possible sequelae of erythrolysis occur such as haemoglobinaemia, haemoglobinuria, icterus or haemosiderosis. These findings strongly imply post-haemorrhagic consequences, and indeed were similar to those resulting from jugular bleeding at the rate of some half litre per day. A marked myeloid hyperplasia of the bone marrow, itself evidence of active erythropoiesis, argues against the possibility of a primary disturbance of the erythropoietic tissues, as also do the striking recovery rates which may occur in the anaemic symptoms. The evidence of anisocytosis, polychromasia, punctate basophilia and the other changes in the blood cells is, therefore, for a secondary change in the blood-forming tissues due to the anaemia. The work strongly favours the view that direct blood loss causes the anaemia of haemonchosis, in which the haematological changes are the consequence of a relative compensatory failure and final exhaustion of the haemopoietic tissues. The single example of haemosiderosis ascribed to haemonchosis is the record of Fraser (1930). Since no worker has obtained any evidence to confirm this we must suppose that Fraser was dealing with another condition of different aetiology present in his worm-infested animals.

Martin and Ross (1935), on the basis of phosphate determinations, made estimates of the blood utilised by reproducing female worms. The calculated loss to the host fell far short of the amount considered essential for production of the observed blood changes. They recognised, however, that their estimate of blood loss was minimal since it allowed neither for male activities, nor for blood ingested "in a spirit of wantonness" in excess of the worms' requirements, and it ignored possible haemorrhage from vacated attachment sites. The probable importance of this last factor was indicated by the observations of Boughton & Hardy (1935) on the frequent changes of site by adult worms on the mucosa of fistulate abomasa. These authors have recorded capillary haemorrhage from newly-quitted attachment sites lasting for as long as 7 minutes. Gastric haemorrhage has also been recorded by Andrews

(1942) and Stoll (1943) who agree that in newly-infected animals it does not occur until the first parasitic moult has been passed, but thereafter it increases progressively with the development of the parasite population. The failure of Stewart (1933) to identify blood in abomasal ingesta of sheep infested with *H. contortus* is surprising.

The only other major contribution to our knowledge of the pathogenesis of anaemia in experimental haemonchosis is that of Andrews (1942) whose findings confirm Fourie's conclusions in all material respects. In addition to studying the development and progress of the anaemic changes in selected examples from his 19 experimental animals Andrews also estimated the blood passed in the faeces of 2 of them. Unfortunately the history of these 2 animals was not typical of haemonchosis, death supervening before the infestation had reached patency. Blood was not identified in measurable quantities until after 10 days from infestation, yet by this time haemoglobin levels, packed red-cell volumes and red-cell counts had already declined drastically. Nevertheless, during the next 10 days or so the total faecal blood was estimated at about 1½ and 2½ litres, quantities calculated as representing from 1½ to 2½ times the total blood volumes of the animals. Jugular bleeding equivalent to 2½ blood volumes removed during a 15-day period from a worm-free animal produced in it an acute anaemia very similar to those of the worm infested animals, all 3 examples terminating at a level of some 2×10^6 red cells per cu. ml.

Both Fourie and Andrews obtained comparable white-cell pictures between infested and bled animals. A moderate neutrophil leucocytosis occurred irrespective of the cause of the anaemia, and there was no real difference between the differential counts of bled animals and fatal cases of haemonchosis. Many recovery cases, on the other hand, exhibited a degree of eosinophilia which is taken by both authors to be connected with the development of resistance to infestation. These results contain no suggestion of a toxic factor but give further support to the opinion that anaemia in haemonchosis is due to direct blood loss.

Against this apparently well established case, Holman and Pattison (1941) have registered a note of dissent. They worked with a group of sheep carrying naturally acquired infestations of which *H. contortus* was a prominent member. Although their findings were not very different from those of Fourie and Andrews they laid great emphasis on the variability they observed in erythrocyte volumes. From this, and from a dubiously permissive argument based on analogy with *Diphyllobothrium latum* infestation they concluded that "in natural worm infestations causing parasitic gastritis the associated anaemia is due to disease of the alimentary tract, and that it is not a definite post-haemorrhagic anaemia," although they admitted the coincidence of the last. They sought to dispute Fourie's conclusions on the grounds that his data did not indicate a megacytic change in all cases and suggested that he also was concerned with anaemias of heterologous aetiology. Our colleague M. R. Brambell has pointed out, however, that Holman and Pattison found increased cell volume to be due to increased "thickness" independently of cell diameters. Moreover, cell volume could increase when diameters were reduced. Fourie's volume indices were estimated from diameter measurements hence the contention that variability in cell volume occurred in his cases is not compatible with the critics' own premise. If we accept the opinion of Holman and Pattison that a dyshaemopoiesis of alimentary origin contributed to the changes in their sheep we are still entitled to regard it as due to the activities of the other worm species rather than of the *H. contortus*. For evidence of a quite different pathogenic mechanism we turn to *T. axei*.

Unlike *H. contortus* which produces the same type of effect in all cases, this species has produced strikingly different results under different circumstances. Doran (1955) observed no significant blood changes in infested sheep. Gibson (1954) obtained a normocytic oligocythaemia developing slowly, some 6 weeks post infection, in poorly nourished lambs but saw no changes in well-nourished ones although these were much younger. Kates and Turner (1960) worked with young lambs in which the infestation terminated fatally at or before 6 weeks post infection, and in these a polycythaemia developed at about 3 weeks and persisted until the end. Finally, Leland *et al.* (1960) described a course in lambs in which an early polycythaemia gradually declined to be succeeded by an oligocythaemia. The same sequence was even more constant in calves (Leland *et al.* 1959). The last-named authors identified the clue which reconciles the apparently contradictory results. They showed that the early polycythaemia was a relative one due to reduction in blood volume consequent upon a disturbance in water balance associated with enteritis and diarrhoea. Then, although the reduction in volume persisted, there occurred a real decline in the red-cell levels so that the animals became anaemic with a reduced blood volume containing fewer red-cells. Kates and Turner's animals died before the second stage was reached, while Gibson's animals developed their symptoms so much less rapidly that the initial relative polycythaemia was not remarkable. As regards the nature of the anaemia, Gibson found no changes in the haematological picture apart from a reduction in the number of cells, there being no evidence of regenerative forms. The bone marrow showed no changes in response to the anaemia, and it was concluded that this was of dyshaemopoietic type due to deprivation of some essential haemopoietic factor or factors.

We have in *T. axei* infestation an example where qualitatively different results are manifest by different degrees of severity of the disease, whereas in haemonchosis quantitative differences only are evident. *A priori* the severity of the disease could vary with (a) the age of the animals, (b) their nutritional status, (c) the pathogenic potential of the parasite strain, and (d) the quantitative features of the infestation. In field experiences the impression is difficult to avoid that younger or more poorly nourished animals suffer more damage from parasitism than do their older or better-filled fellows. This

is often the mere consequence of more or fewer parasites, older and better nourished animals being more resistant to the establishment of parasitic populations. Although his groups were insufficiently large to provide a conclusive result, Lucker (1953) working with *H. contortus* in lambs of 3 and 8 weeks was able to suggest that "capacity to combat the effects of parasitism may tend to increase with age." Gibson (1954 & 1954a) examined the effect of *T. axei* on 3-month-old lambs on a high, and 8-month-old lambs on a low, plane of nutrition; but his groups also were too small to permit definitive conclusions. He found that the high plane of nutrition allowed the animals to escape the dyshaemopoietic anaemia suffered by those on the low nutritional plane in spite of the age disadvantage, and concluded that the anaemia was prevented by the better feeding. But the low plane lambs carried a much heavier worm burden at death, and it appears, therefore, equally valid to suggest that the higher plane of nutrition enhanced resistance to parasite establishment which consequently reduced the infestations and hence their pathogenicity.

Strain variation within helminth species has become a subject of increasing interest in recent years. Roberts *et al.* (1954) examined the statistical distribution of morphological variations in *H. contortus* populations in Queensland. The relatively constant differences between strains from sheep and cattle led them to elevate the cattle strain to specific status. The cytological differences recognised by Bremner (1955) in material from the same region support this view. In the United States the picture appears to be more confused since Drudge *et al.* (1957, 1959) in Kentucky, and Madsen and Whitlock (1958) in New York have described strains of sheep origin significantly divergent from, and intermediate between, the 2 Queensland forms. Drudge *et al.* have identified morphologically inseparable strains of this intermediate type which exhibit marked differences in phenothiazine tolerance. Allen *et al.* (1958) appear to have recorded the only example so far of a possible strain difference in pathogenicity within the *Haemonchus* complex: they consider that a strain, within the statistical range of the Queensland cattle strain, isolated from feral sheep was less pathogenic than strains of domestic origin in domestic New Mexico sheep. The differences observed in growth and haemoglobin levels could, however, have been a function of the numerical differences in the established populations implied but not specified by the authors. As regards *T. axei*, Kates and Turner (1960), from a brief review of the published data on strains of ovine, bovine and equine origin, consider that pathogenic differences between them are insignificant. Drudge *et al.* (1959) and Leland *et al.* (1960), on the other hand, suggest differently: the first authors concluded that a strain of equine origin was more pathogenic to calves than other strains, while the second considered that an equine (presumably the same) strain was less pathogenic to lambs than other strains. We must accept that the question of differences in pathogenicity, although still wide open, has a potentially important part to play in problems of quantitation to which we now turn.

There are 3 practicable measures which can be used to quantify the worm populations of experimentally infested animals, namely: (1) counts of larvae administered. Mayhew (1948) is alone in his view that assessment of infective doses is too inaccurate for practicability); (2) counts of eggs passed and (3) counts of adults and larvae present at death or slaughter; and these methods obviously have considerably greater value when used together than when used singly. For example, the quantitative value of Fourie's work is impaired by omission of information on all but the first, while Mayhew's many publications suffer from his neglect to use this measure.

Very few attempts have been made to relate changes in the blood picture to any of these criteria, partly, perhaps, on account of the difficulty in experimental work, of obtaining groups containing sufficient numbers to overcome the statistical bogy of individual variation. Where larger groups have been examined under field conditions some workers have obtained relationships between egg-counts and haematocrit or haemoglobin levels. For example, Hawkins *et al.* (1944) observed an inverse relationship between rise and fall of egg-count and haemoglobin in lambs during the course of their acquisition of, and recovery from, mixed infestations containing *H. contortus*. Grunsell (1955) obtained a significant negative regression of haematocrits on log egg counts in hill ewes in late winter and spring when infestation by *Ostertagia* spp. and *Trichostrongylus* spp. was at its highest level of the year. Whitlock (1958) has published a scatter diagram between the maximum haematocrit fall and the square root of the maximum egg count observed in individual sheep during 2 grazing seasons on an area where *H. contortus* was a prominent member of the gut-worm complex. Although it was not his concern to demonstrate it the data contain a significant rectilinear regression of the one datum on the other. Field *et al.* (1960) found a significant negative correlation during the spring rise period in housed ewes between haematocrits and log egg counts, while the present authors (unpublished) have observed the same in housed hoggs. In both these cases *H. contortus* was the dominant member of the infestation.

Egg-counts serve only to quantify adult female reproductive activity. Now although the requirements for egg production will make heavy nutritional demands upon the host and will presumably in turn contribute largely to the pathogenic effects, it is conceivable that the other stages of the life-cycle will also contribute their quota. In particular we might expect the developing larva to cause some damage. Indeed there is much evidence to indicate that larvae can cause damage, and in *Haemonchus* infections that they also cause haemorrhage and produce anaemia. Falls in red-cell levels during the prepatent infective period have been described by Andrews (1942), Delaune and Mayhew (1943 and Shumard *et al.* (1957) among others. Fourie, on the other hand, did not find such effects: anaemia did not become evident in his animals until after patency had occurred. There seems little doubt that these differences are related to the numbers of larvae

undergoing metamorphosis at the same time, but quantitative assessment of the larval factor contains additional problems for the experimentalist.

First there is the choice between administration of a given number of larvae on a single occasion and serial dosing over a period of days or weeks. The single large dose is an improbable event in nature while serial dosing may be complicated by the possible development of immunity. In either case 3 possibilities may be envisaged for the individual larva, namely: (1) that it exsheaths and proceeds forthwith to complete its development to the adult stage; (2) that it exsheaths but its further development is arrested on account either of the natural resistance of inexperienced or the acquired resistance of experienced hosts, and (3) that it is passed straight through the host with or without undergoing exsheathment. In susceptible animals a large proportion enjoy the first fate, and under these circumstances provided the number is large enough significant host effects may occur during the prepatent period. Non-experienced hosts, however, vary considerably in their susceptibility and this modifies the proportion which develops directly, but as to the proportions which fall into the other 2 categories we can only speculate. The mode of administration may affect the result, as Tetley (1959) suggests. An oral dose in aqueous suspension may go into the rumen or abomasum direct and this may affect the exsheathment. To ensure entry to the rumen, where it is probable that exsheathment of stomach worms mainly occurs, Tetley used filter-paper, but unfortunately his experimental design did not admit comparison between fluid and filter-paper dosing. The establishment from filter-paper doses, however, appeared to be as variable as that from fluid doses.

There is no reason to suppose that the proportion of a larval ingest which develops directly is constant in any given circumstance, and it appears rather that this proportion decreases with increasing larval challenges. Kates and Turner (1960) have even identified an absolute decrease in the numbers of *T. axei* developing from increased larval doses. It may be remarked *pari passu* that the relationship which they dismiss as an approximately inverse one is, in fact, highly significant: the correlation coefficient calculated from their data between the logarithm of the infective dose and actual establishment approaches unity. It would appear that establishment in non-immune animals is subject to a density dependant factor, and should this prove general the quantitation of larval effects will prove more obdurate than ever.

In view of the significant correlations between egg-count and haematocrits that can be obtained with *Haemonchus* infestations there is a possibility of the recognition of larval effects by more recondite statistical methods. Campbell *et al.* (1960) have studied the haematocrits in 3 groups of 8, nine-month-old lambs infected at logarithmically spaced rates of 10, 100 and 1,000 larvae per day. Significant correlations were obtained between the egg counts expressed as logarithms and the fall in packed red-cell volume. After adjustment for the differences associated with the egg count, the group means of the packed-cell volumes were not significantly different. It is concluded that the effect of the larvae must in these circumstances have been insignificant, in comparison to that of reproducing adults, since in spite of the large group differences in the number of larvae received, there was no recognisable difference in the effects that might have been attributed to them. The problem requires further examination under other circumstances but the present findings on non-fatal infestations provide grounds to dispute as a generalisation the view that larval development in this species is a significant factor in the genesis of disease symptoms in the host.

The statistical relationship between haematocrit fall and egg count argues further in favour of the post-haemorrhagic nature of the anaemia in non-fatal haemonchosis. Where the disease reaches fatal proportions, however, we must expect that the rectilinear relationship will cease to hold. As the haemopoietic tissues approach exhaustion death will supervene without direct reference to numbers.

In anaemias whose aetiology is different, the quantitative relationship with the casual infestation will almost certainly be different from that described so far for *H. contortus*, but the work on these awaits an undertaking and until then speculation is fruitless.

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THE SPEAKERS' INTRODUCTION

The Chairman, Mr. A. S. Chapman, said that the theme of the Congress seemed to be economics of animal health and since the economics of farming was more than ever linked up with sheep, and they played a great part in Scottish agriculture, the papers about to be discussed were very timely. Dr. Montgomerie was so well known that any formal introduction could be dispensed with.

Dr. Montgomerie said that he had particular reason for expressing thanks to the Provisional Committee. His first contribution to a Veterinary Congress was made in Aberdeen in 1924. After 36 years it would seem usual to look upon the individual as one who had got into the backwaters. Therefore he was particularly appreciative of the invitation to take part in this symposium as an indication that he had not yet reached that stage of decline.

Over that period there had been tremendous advances in the sphere of the control of sheep diseases, and it was opportune that they should be meeting in Glasgow, where Professor Gager laid the foundation stone for much that had been achieved since.

His subject was relatively simple and its compass was relatively restricted. However, attention should be drawn to that section of Dr. Watt's paper which said:

> "The most striking disease is, of course, pulpy kidney disease or enterotoxaemia which is undoubtedly a disease encouraged by modern methods of intensive farming. This disease occurs at most times of the year in one class of sheep or another and the overall losses must be heavy."

Dr. Montgomerie had taken that conception as the text, in effect, for his own small paper. Pulpy kidney disease could not be regarded as an infectious or contagious disease according to the normal usage of those terms; it must be regarded as a disease of the animal's environment. It was associated particularly with modern methods of economic husbandry. These methods seemed to have one great objective: to make an animal grow twice as fast as previously and to fatten at about three times the previous rate.

Perhaps Dr. Watt would forgive another quotation from his paper:

> "As, however, the only really effective prophylactic measure to control pasteurellosis of this type is to administer a severe check to the animals by immediately moving them to a bare pasture or confining them for part of the day, the question is of more academic than practical importance as this treatment will also control enterotoxaemia."

The veterinary profession had no right to adopt that type of method for control of disease if it defeated the objective of modern methods of management. There were modern methods which introduced serious disease risks which existed to only a small extent when the methods of the past were used. But it was not their job to encourage people to slip into the methods of the past, but surely to meet the challenge—a serious one, whether they thought of broiler chickens or the rapid fattening of animals to get maximum production. It was up to the profession not to preach against modern methods for maximum production but to set about finding the ways and means by which these methods could be employed without the awful fear of disease intervening.

Pulpy kidney disease was caused by the multiplication in the intestine of an organism which was not uncommonly there in the normal way. The greater the extent to which they forced animals for increased production, the greater the extent to which that organism found conditions in the intestine suitable for toxin production. That could be met by one way only: by creating an immune population. Under modern conditions of sheep management the only line to take was to keep the population immune.

With the advances that had been made in the quality of vaccines against *Cl. welchii*, they had in their hands the tools to do the job. The question was how they could best be employed, in the varying circumstances, to keep the flock immune.

The suggestion of flock immunity was not new. In human medicine it had been undertaken for many years so far as smallpox was concerned. They had seen the success of the diphtheria immunisation campaign.

He did want to pay a tribute to the V.I.O. service which had done so much to give a better understanding of the disease position. It had been shown that pulpy kidney disease was a serious factor in quite young lambs on hill farms and he would be interested to hear the views of Investigation Officers who had to face this problem.

Dr. Karol S. Chodnik (Greenford) said that he supported wholeheartedly most of what Dr. Montgomerie had said, since it confirmed many of the recommendations now in current use. It was, however, an opinion of his and his colleagues, that a single dose given to pregnant ewes previously vaccinated was adequate and satisfactory. In December of 1957, 24 ewes, vaccinated a year before, were sampled and they were all found to be fully protected. They received a single dose and when sampled a year later were shown to be again immune. The same result was obtained in a number of animals vaccinated and kept under strict experimental confinement for the purpose of direct challenge. The contention was also borne out from field observation both in this country and in New Zealand. In recommending single dose injection, it was stressed that in pregnant ewes the injection was primarily for the protection of the newborn lamb by transferred immunity. If the injection of adsorbed toxoid was given too close to lambing the full benefit might not result. Undoubtedly the first injection was primarily a sensitising dose, but in lambs born to previously vaccinated ewes, the drop in the antitoxin level was delayed by a single injection to such an extent that the procedure was a practical proposition for early fat lambs.

Dr. Montgomerie said he did not think that 24 ewes could give results on which to base a strong recommendation. Over the years he had seen the better responses with the improved vaccines. To allow a year to pass since the previous injection was, in his opinion, running a risk.

The Chairman asked Dr. Watt to introduce his paper.

Dr. Watt said it was a disadvantage to follow Dr. Montgomerie who could always carry off these occasions with inimitable skill. A paper dealing with sudden death in sheep must, of necessity, cover only a very small portion of the field related to those two words. No other animals of the domestic variety were so liable to be found dead as sheep. That was due partly to their extreme susceptibility to change. Faulty observation often led to death being reported as sudden when in fact the animal had been ill. Even an animal that was quite ill would appear to be fairly normal when the shepherd and his dog appeared in the field.

The first essential in controlling the disease was proper diagnosis and that was not as easy as one might think. Pulpy kidney disease had to a large extent taken over from braxy, but there were other killers and a number of causes of sudden death were listed at the end of the paper. There were, however, a number of conditions which did not fall into any category. Diagnosis was dependent on accurate history and the interpretation of the history was then dependent on *post-mortem* findings. Full history was extremely difficult to elicit. It was more important than *post-mortem* examination, but most of the information which was volunteered when a note was sent to a laboratory consisted of irrelevant details. The best way was by question and answer. The phrase "sudden death" itself meant different things to different people. It was sometimes ascertained in questioning people that an animal had died suddenly—after being ill for a fortnight. Sheep diseases and deaths were so seasonal that if there were a full *post-mortem* examination in all cases the job would be quite unmanageable.

The paper attempted to classify according to ages and times of the year. The diseases dealt with did not make up an exhaustive list. To introduce all the exceptions would make the picture confusing. Intensive management in experimental laboratories had led to the appearance of conditions rarely, if ever, seen in the field diseases, but they could, however, appear if the practice of housing sheep and wintering hoggs in sheds were extended. An example of that was the cardiac myopathy described in experimental sheep at Moredun by Nisbet.

Management factors undoubtedly played the major part in the production of diseases characterised by sudden death, as undoubtedly they did in other conditions. It was essential that, while every effort was made to protect the flocks by vaccines and sera, the tremendous importance of managemental causes was not overlooked. Flock management should be one of the weapons in the armoury of the veterinary surgeon, if not in fact the most important weapon.

But they must face the fact that a proportion of sudden deaths could not readily be ascribed to any definite disease process, and *post-mortem* examination still too frequently ended without a definite conclusion being reached. It was in such cases that control by change in management was the most productive line in prophylaxis. It was essential, therefore, that the veterinary surgeon be thoroughly conversant with the systems of management adopted in the area in which he practised. It was necessary not only to prescribe the desirable change in management; the measures suggested must be practical.

When deaths could definitely be pinned down to pulpy kidney disease prophylaxis was, of course, relatively simple. It should not be forgotten, however, that the conditions conducive to pulpy kidney gave predisposition to other things, such as acute pasteurellosis. The value of vaccination in such cases was difficult to assess as the very fact that the animals were subjected to a check by the operation could lead to a cessation in the trouble.

The most difficult problems in differential diagnosis were the metabolic diseases and pulpy kidney disease. As the methods of control were so very different accuracy of diagnosis here was of the greatest importance. The list of diseases at the end of the paper did not reflect their true economic importance. Enterotoxaemia was the most widespread and the losses from it must be very considerable. The trouble could be controlled by efficient vaccines. Perhaps the most disturbing factor relating to the list was the increase in hypomagnesaemia, which in bad years caused losses which could be very severe on individual farms.

One should not be misled into diagnosing pulpy kidney disease merely on the presence of pulpy kidney toxins in the gut as these could be present in adequately protected sheep dying of acute pasteurellosis.

Mr. Box (Greenford) asked whether there was any reliable means of diagnosing death due to pulpy kidney if pulpy kidney toxin in the gut did not necessarily mean that death had been caused by it.

Dr. Watt said that his routine was to decide, having in mind the history, whether the disease was likely and whether the animal was particularly susceptible; also, whether the *post-mortem* picture suggested an acute anaerobic condition. He had tried to demonstrate sugar in the urine but an animal that had died of pulpy kidney seldom had any urine in the bladder. He also used biological demonstration of toxins and the smear from the small intestine to show large numbers of *Cl. welchii* present in the bowel, but did not go further than that.

Dr. J. D. Rankin (Compton) asked whether sheep died of anthrax.

Dr. Watt said that sheep were as susceptible to anthrax as were cattle.

Mr. Roberts said that he fully agreed with Dr. Montgomerie's point that the best way to control pulpy kidney was by vaccination, and it was unnecessary for the manufacturers of vaccines to preach very much about that. Practitioners and farmers accepted that vaccines were quite efficient in controlling such diseases. He had become executor of an estate in which there were 200 ewes. Everything was done to control pulpy kidney diseases, but when the spring came there were a lot of deaths. Undoubtedly sudden deaths did occur.

So far as pulpy kidney disease was concerned the problem was confined to that system of sheep husbandry by which hill sheep were brought down to very much better pasture. Next, they had to tackle hypomagnesaemia, and they might advise the shepherd to go around with a syringe. But while one sheep was being attended to, another was dying a mile away. The difficulty of making a diagnosis in some cases was more difficult than had been suggested.

It was unfortunate that the word "pasteurellosis" was being used. He would be delighted if anybody in the country could present him with a pathogenic strain of *Pasteurella* which had been associated with the disease in sheep.

Dr. Watt said he felt he should say he had endeavoured in the paper to illustrate the *post-mortem* findings with typical cases. What one considered a typical case and what the text-book considered a typical case could be different. If the history was borne in mind the possibilities could be narrowed down considerably. On the question of nomenclature, the matter went back to the bacteriologist who classified the *Pasteurella*, and he could not think of a better term than "pasteurellosis" so long as the associated organism was classified as *Pasteurella haemolytica*.

The Chairman asked Dr. Mackay to introduce the paper.

Dr. Mackay began by thanking the Provisional Committee for asking him and his co-authors to present a paper to the Congress.

Slide: Table I summarised the origin of the various materials used. He was glad of the opportunity to thank Dr. Gordon and Mr. Pattison who provided the goat passage material. The apparent change in behaviour after passage through goats was suggestive evidence of a living agent.

It was also quite clear that not only were there differences between sheep and goats in respect of the experimental disease but also between one breed of sheep and another. In view of the variable frequency of takes in similar experiments with the same breed of sheep and also of differences between breeds, it is necessary for a critical interpretation to have more information on factors which affect variations in incidence. Factors such as previous management and genetical constitution may be important. It is therefore desirable to have control of a breeding flock in order, not only to provide more carefully balanced groups for inoculation, but also to study genetical aspects. In addition, an experimental breeding flock is necessary in order to relate observations on induced scrapie to those on the natural disease.

With these ends in view a flock was started in 1955. Suffolks were chosen because the contacts that had been built up with their breeders gave more prospect of yielding a sufficiently high frequency of natural cases to make study worth while. At that stage it was assumed that a practical incidence of experimental scrapie would be able to be induced in this stock. However as they had mentioned in the discussion of the present paper the inoculation of their Cheviot pool material into 6-month old Suffolk sheep did not give rise to scrapie within a 12-month period, and this was so whether the sheep inoculated were born from scrapied or non-scrapied parents.

The need to use sheep of known genetic background and of proven susceptibility to both the natural and experimental disease is self evident, but it will be equally apparent from our results that this may not be easy to achieve.

Slide: Table III summarised the results of boiling and autoclaving scrapie tissue suspensions prior to inoculation. In addition to the results reported in the paper it would be seen that one case of scrapie had developed in a group of 6 goats given boiled scrapie sheep brain. This case occurred at 38 months following inoculation. (See Table III opposite.)

Another recent finding shown in the Table was that supernates of scrapie sheep spleen suspensions were still capable of inducing the disease after boiling. There were thus no grounds for suspecting that the apparent heat resistance of the agent was due to a protective effect of brain tissue.

Scrapie had been called a research worker's disease. He believed that he and his colleagues would want to study the

disease even if it had no economic importance because of its intrinsic interest, but it was possibly of greater economic importance than the majority of veterinary surgeons realised. He hoped that some of those present who had particular interest in sheep practice would have something to say on the incidence of the disease in their areas.

Mr. J. C. Wood (Berkhamsted) said that several years ago he bought the first prize pen of shearling ewes at a certain breed society's annual sale. Within the following 3 lambing seasons 7 out of 10 were lost through scrapie. That was mentioned merely as an example of the possible economic importance of the disease.

Mr. Roberts asked why Welsh mountain sheep could not be used in connexion with control.

Dr. Mackay said there did not seem necessarily to be a close relationship between susceptibility to the experimental disease and susceptibility to the natural disease. Mr. Pattison had injected 30 Welsh mountain sheep with Cheviot material and only 1 case developed 8 months after inoculation.

The Chairman asked Mr. A. C. Gardiner to present the next paper and said its importance needed no emphasising.

Mr. Gardiner said that, as a chemist recently recruited to a parasitological laboratory, it was with some alarm that he addressed a veterinary audience on a veterinary topic. The alarm was increased by the fact that Dr. Campbell, who was the expert behind the paper was on the Continent and would not be at hand to help him out of deep water.

The paper gave a brief account of anaemias associated with internal parasites. Parasitic anaemias could be due to 3 main mechanisms, either acting singly or in any combination. These were (a) copious blood loss due to simple haemorrhage, (b) disturbance in the blood forming mechanisms by the parasite and (c) destruction of blood cells by haemolytic agents.

In the case of experimental infestations with *Trichostrongylus axei* there was an initial decrease in blood volume due to enteritis and diarrhoea and this was followed as the infestation progressed by a true reduction in the numbers of circulating blood cells. Depending on the numbers of larvae ingested and on the rate of ingestion the two mechanisms interacted to produce widely differing effects. Gibson's experiments with *T. axei* infestations produced no evidence of haemolytic changes and his conclusions from experiments carried out on different nutritional planes indicated a dishaemopoietic anaemia.

With regard to *Haemonchus contortus* infestations, the differences produced in the blood picture by varying severities of attack, were more or less quantitative. Qualitative changes did occur however when the anaemia was very advanced. The progress of an infestation and anaemia as followed by changes in faecal egg output and haematocrit values, was demonstrated with a slide. Another slide showed a graph correlating haemoglobin levels and packed cell volumes as an indication of the normality of cells despite the great reduction in numbers.

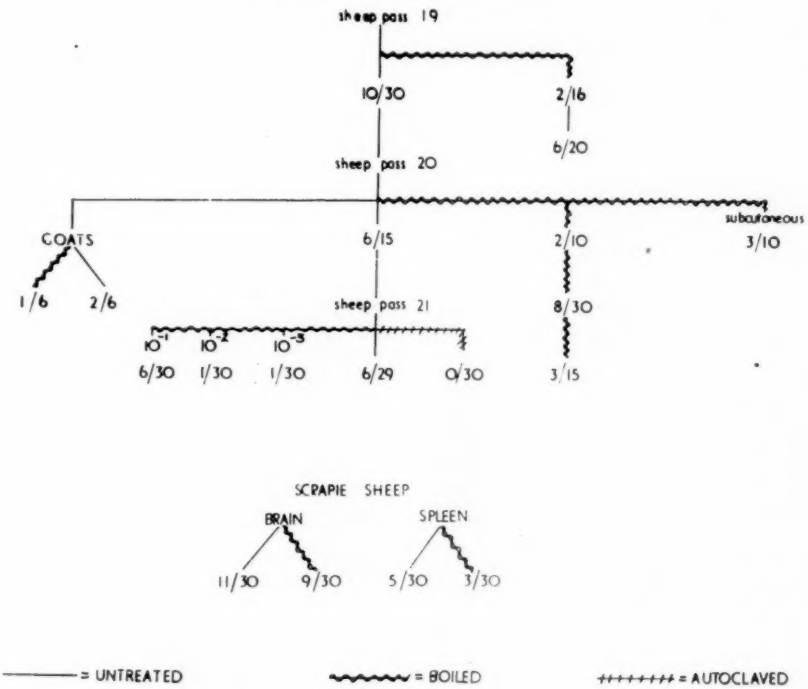
The foregoing facts referred in the main to experimental infestations. In the field the interplay of many other factors such as disease, inadequate feeding, climatic conditions and parasitism with other species made the overall picture exceedingly complex.

Mr. Gardiner said that he did not know of much work of this kind but radioactive tracers would obviously be very useful in this field. The difficulty to start with would be in getting the necessary equipment to deal with radioactive materials. However, it had been worked out by normal methods that a severe anaemia could be produced by removing blood to the extent of 1½ to 2½ times the normal blood volume of the sheep if this were done over a short period. The time factor though would obviously make a difference to the severity of the anaemia produced.

The General Discussion

Dr. Mulligan (Glasgow) said it had been interesting to see from the paper that we are still in considerable ignorance as to which parasites consume blood, and as to the amount consumed by those that did. It was strange that more work had not been done in that field using isotopically

DR. J. M. K. MACKAY.—TABLE III



labelled red cells, to obtain an estimate of the blood being lost. It was possible to inject labelled cells into an animal; after a time, when the animal was killed, a count and an estimate could be made. It would be quite appropriate to use sheep which carried a mixed burden of parasites. Had much been done along those lines so far?

Mr. Gardiner said he did not know of much work of that kind taking place. A difficulty was equipment and knowledge of how to handle radioactive equipment and material. It had been worked out that to produce a fairly severe anaemia the worms must cause the sheep to lose between 1½ and 2½ times its normal blood volume. But that depended a lot on the time factor. Obviously, the quicker it happened the less there would have to be removed.

Dr. G. R. Smith (Moredun) said he would like to support what was said earlier by Mr. Roberts concerning the term "pasteurellosis" as applied to sheep disease. It led to confusion because it made most people think immediately of *Pasteurella septica*, a rare cause of disease in sheep in this country. The organism most commonly associated with pneumonia and also causing septicaemic infections of sheep was in fact *Pasteurella haemolytica*. This organism had probably been unwisely classed in the genus *Pasteurella* and several workers had preferred to give it the less misleading name "haemolytic coccobacillus."

Dr. J. T. Stamp (Moredun) referred to the point raised about the use of Welsh mountain sheep in scrapie research. The essential difficulty in scrapie research was the transmission of the replicating agent after boiling. Always at the back of one's mind is the thought that things might be happening which are not appreciated as, for example, the stimulation of a latent scrapie agent and one would therefore need to be very sure that Welsh mountain sheep do not take natural scrapie. There are various reports that some sheep of this breed do suffer from scrapie which makes one suspicious of using them since it would be unlikely that they would prove a more satisfactory breed than, say, the Scottish black-faced mountain sheep which have been used extensively in scrapie research. If scrapie does not occur commonly in a breed can it be argued that it is widely present but the disease does not show itself clinically because of some environmental factor?

Dr. Mackay said that the idea of stimulation had occupied much thought but there were no actual results to show that the thing which withstood boiling was not multiplying. It was likely that they were dealing with a factor which withstood boiling. Fresh hope in this connexion came from Bachrach's* recent work on foot-and-mouth disease. If free virus was boiled for 5 minutes the resultant product was not infective, but if one took the boiled material and extracted the RNA this material was in fact capable of producing disease either in a tissue culture or in an animal.

Mr. K. W. Aspinall (Nyasaland) said it had been mentioned that toxins were found in the bowel of immunised animals, so one could not immunise animals as a diagnostic factor. If an animal were immunised against *Clostridia*, would it not fight the toxins in the bowel?

Dr. Watt said he believed that by immunising an animal one would not to any real extent interfere with the flora that was in the intestine and not attacking the gut. So he could not see its being used to eliminate the organism or to neutralise toxin in the lumen of the intestine.

Dr. Montgomerie asked why there should not be toxin in the gut of an immunised animal. *Cl. welchii* type D was a common inhabitant of the gut and constantly multiplied to a variable extent, depending on the suitability of the contents as a growth medium. Why then should not it so happen that an animal died of another disease at a time when there was a phase of material multiplication going on and quite a level of toxin in the intestine? Could it not be accepted that stasis of the intestine arose as an effect of the other disease and this, in itself, set the stage for multiplication and toxin production in the intestine, whether or no the sheep was highly immune?

Dr. J. D. Rankin (Compton) said he was not working on scrapie himself and apologised if he was about to make a wrong supposition. However, it was his impression that Mr. Pattison had inoculated many goats, probably 70 or 90, with scrapie material from sheep or goats by numerous routes and had never failed to cause scrapie in goats. It appeared that in Mr. Mackay's experiments not 100 per cent. of the goats were infected. Could that discrepancy be explained?

Dr. Mackay said that he had asked Mr. Pattison the same question. But, so far as he was aware, if scrapie goat brain was used and the injection was given intracerebrally the take was always 100 per cent. It must be that in the experiments described, using scrapied sheep brain as source, that the animals have simply not been under experiment long enough (38 months) and that the remaining goats in each group will eventually go down.

The Chairman said that it only remained for him to thank the authors and the speakers who had contributed to the discussion. They were indebted to all those who had made the meeting a success.

The vote of thanks was carried with applause and the meeting then concluded.

* Bachrach, H. L. (1959). Biochemical and Biophysical Research Communications, **1**, 356.

Symposium on Sheep Diseases.—*Concluded.*

Surgical Interference in Dilatation of the Stomach of the Dog (Torsion of the Stomach)

BY

J. B. TUTT and T. G. YARROW

Winchester

Introduction

AN excellent article on the condition and the morbid anatomy has already been published by Blackburn and McFarlane (1944). It is not proposed to enlarge upon their findings other than to give a brief description of the condition which is most commonly seen in the large breeds of dogs—Great Danes, Bloodhounds and Red Setters.

The condition is sudden in onset, the symptoms usually commencing soon after a meal and giving rise to rapid abdominal swelling and pain. The animal is reluctant to move and will only move with care. Percussion of the distended abdomen reveals a tympanitic sound and the animal appears shocked. Unless the case is treated with extreme urgency, surgical intervention will be too late and death may occur in a few hours.

Case History

Subject. A 3-year-old Irish Wolf Hound. Height, 33 inches; weight, 120 lb., approximately.

History and symptoms. The animal had eaten a full meal at 4.30 p.m. and was found to be in a distressed condition at 7.15 p.m. The dog was in pain with obvious abdominal distension due to tympany, and its temperature was 103° F. A diagnosis of torsion of the stomach with dilatation was made, and arrangements for surgical intervention were made immediately.

Preparation and anaesthesia. Chlorpromazine* intravenously was given as a premedicant together with atropine sulphate 1/100 gr. Anaesthesia was

* Largactil: May & Baker, Ltd.

induced with pentobarbitone sodium* given intravenously. The chlorpromazine premedication and the shocked condition of the animal combined to reduce the amount of pentobarbitone sodium necessary for anaesthesia, and only 12 gr. were used. The authors would have preferred to have used halothane as being much safer in such a shocked animal, but it was not available.

Surgical Approach. A left paracostal incision was made about 1 inch away and approximately 8 inches long down through the skin and abdominal muscles into the abdominal cavity. The stomach was immediately seen to be grossly distended and very dark in colour. The spleen lay in the left umbilical and pubic region; it was enormously distended and black in colour.

A trochar puncture, using a large-bore hypodermic needle, was made in the stomach to release the distension and to obtain the following two objectives: (1) to release the immediate danger of asphyxia, and (2) to enable the stomach to be manipulated easily.

The spleen was then brought out of the abdominal cavity; the splenic vessels were engorged and passed dorsally over the distended stomach.

It was then decided to open the stomach and to remove the contents, again to facilitate handling. This was done by making a small incision, and the contents were squeezed out into a bowl. About 4 pints of material were removed in this way and the gastric incision was closed in the usual manner.

It was extremely difficult to tell which way the stomach had rotated. Great difficulty was experienced in locating the greater curvature and the pylorus, but the latter was eventually found on the right side of the body. The stomach was rotated and eventually both the pylorus and the oesophagus could be seen and it was evident that the torsion had been reduced. It must be admitted that at no time during the first stages of the operation were the operators certain in which direction the torsion had occurred. Once the pylorus had been located and brought into its correct position, the position of the stomach became normal. It was definitely the result of trial and error, but in the author's view the twist was anti-clockwise.

The stomach was then fixed in position by a thread suture to the ventral peritoneum, close to the anterior inner aspect of the incision. Internal oblique muscles, transversalis muscles and peritoneum were closed by interrupted thread sutures, and the skin and external oblique muscles were closed by interrupted monofilament nylon suture No. 6. The abdominal cavity had been dusted with crystalline penicillin and the line of the incision was treated with penicillin/dihydrostreptomycin ointment.† A gauze pad and a 4-inch adhesive bandage‡ was then applied. The dog was given 900,000 units of procaine penicillin intramuscularly and, to combat shock, 125 c.c. dextran intravenously.§

The whole surgical procedure from start (when everything had to be got ready) to finish took 2½ hours.

Recovery from anaesthesia was uneventful and the animal was free from narcotic excitement. The next day the dog appeared to be well but was still tranquillised from the chlorpromazine.

After treatment. The dog was given 900,000 units of procaine penicillin for 3 days, together with streptomycin sulphate sulphaguanidine* which was also administered daily for 3 days. On the first day glucose and water in small but repeated amounts with protein hydrolysate only were allowed. On the second and third day a very small amount of solid food was permitted. There was no further distension and gradually the amount of solid food was increased. The animal appeared normal on the second day and continued to progress well. No diarrhoea occurred, and the dog made an uneventful recovery.

Comments

(1) The symptoms make diagnosis easy.

(2) The animal becomes shocked extremely quickly, and for the operation to be successful the case must be treated with extreme urgency. The animal in the case reported above had eaten a meal at 4.30 p.m. and the abdomen was opened at 9 p.m.; even so, the stomach was almost black, the spleen was enlarged and black in colour, and the animal was badly shocked.

(3) Removal of the stomach contents is essential to enable manipulation of the stomach to take place.

(4) The skin incision must be adequate to allow plenty of room for observation and manipulation.

(5) Antibiotic and shock therapy are essential.

A similar case of successful surgical intervention has been described by Wynn Jones (1952).

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* Nembutal.
† Streptopen: Glaxo Laboratories Ltd.
‡ Elastoplast.
§ Intradex; Glaxo Laboratories Ltd.

* Entavet; Allen & Hanbury.

R.A.V.C. PROMOTIONS

The following promotions have been made in the Royal Army Veterinary Corps:—

Lt.-Col. D. F. G. Smith is promoted Colonel; Major F. H. Orr is promoted Lieut.-Colonel, as is Major J. H. Wilkins Major (Veterinary) P. W. Dean of the Royal Horse Guards is promoted to Lieut.-Colonel (Veterinary).

LIVESTOCK IN AFGHANISTAN

There are at present only 10 Government veterinarians in Afghanistan to control animal disease among an estimated 25,000,000 livestock in that country. Even so, the Afghanistan Government, with the advice and help of veterinarians from the Food and Agriculture Organisation, has made so much progress in the control of disease that no widespread epidemics have occurred in Afghanistan for the past 3 years. This is a remarkable achievement in a country which is nearly 3 times as large as the United Kingdom, and where the livestock population is scattered over vast plains and mountain ranges.

Treatment of Bovine Cutaneous Papillomata with Autogenous Vaccine

BY

WALLACE F. BUTLER

Upper Langford, Nr. Bristol

SUMMARY.—A small-scale controlled clinical trial using autogenous vaccine is described.

The results seem to indicate the value of the method which is simple to apply.

A single dose is effective; 2 g. or more of tissue per 60 ml. of glycerol-saline are used.

The aetiology is discussed.

Introduction

WARTS in cattle do not constitute a major problem of disease but occasionally they may become very unsightly and may interfere with the general health of the animal. Moreover, they may sometimes spoil an animal for milk production where an extensive affection of the belly includes the udder.

An opportunity presented itself earlier this year to try vaccination of some of the author's own calves with an autogenous vaccine as a method of treatment for cutaneous papillomata. Since Pearson, Kerr, McCartney and Steele (1958) reported the successful use of vaccination in Ireland, it was decided to use their method.

Material

The animals fell into two groups. Group A had been housed since November, 1959, and prior to this had grazed a permanent pasture with Group B. Group B had remained out-of-doors throughout the winter and out of direct contact with Group A. Table I gives further data on the animals.

TABLE I

DATA ON ALL ANIMALS IN THE GROUPS, WHETHER AFFECTED (+) OR NOT (−).

Group	Identity	Sex	Affected or not	Treated or control	Breed	
		F	—		Angus	×
A	43	F	+	Control	Angus	×
	41	M	+	Treated	Hereford	×
	50	M	+	Control	Hereford	×
	49	M	+	Treated	Hereford	×
		M	—		Hereford	×
B		M	—		Hereford	×
		M	—		Hereford	×
	45	F	+	Control	Friesian	
	44	F	+	Treated	Friesian	

The warts varied in size from about 1 cm. to 3 cm. in diameter, the majority being in the lower size range. They occurred only on the head and neck of the animals, and were clean and "young-looking." There were no obvious signs of secondary infection.

Methods

The method described by Pearson *et al.* (1958) was used, though slightly modified, to prepare the vaccine.

Because the warts were mostly small, only 2 g. of tissue were taken from each affected animal, making a total of 12 grammes. Scissors were used to take the tissue which was kept in a vacuum flask with ice. The next morning the material was homogenised, by means of a Waring Blendor*, in 360 ml. of 50 per cent. glycerol-saline. After allowing the material to settle, the supernatant was decanted and kept at 5° C. for 24 hours. It was then decided to divide the fluid into 2 portions, leaving one at room temperature (ca. 20° C.) and the other at 5° C. for 3 days. After this period, the coarse material, mainly stratum corneum, was removed by filtering through muslin, and the vaccine was left for 48 hours.

In the first trial, vaccine which had been kept at 5° C. for 7 days was used, the controls receiving 50 per cent. glycerol-saline. In the second trial, vaccine which had been kept at 5° C. for 7 days followed by room temperature for 5 days was used on the control animals from the first trial.

The material was injected subcutaneously behind the shoulder in doses of 10 ml. By accident, animal 41 only received 3 ml.

Results

Trial 1. On March 20th, 1960, 8 days after vaccination, there was no observable change in any animal, but on March 27th, 15 days after vaccination, the following results were seen:—

No. 41, no change.

No. 49, regression of lesions. Some very pedunculated, and others shrunken and black.

No. 44, complete regression. No visible or palpable lesions.

Controls, no change.

Trial 2. On March 27th, 3 weeks after the material had been collected, the vaccine which had been kept at room temperature all the time was used in 10-ml. doses on the 3 control animals of Trial 1. These still had warts and they were now larger than at the start of the first trial.

On April 26th, the results were as follows:—

No. 43, warts reduced in number by about 75 per cent.

Nos. 45 and 50, lesions not visible and only palpable with difficulty.

No. 41, lesions still visible but shrivelled and fewer in number.

One month later all the lesions had gone, including those of No. 41.

Discussion

The above results support the findings of other workers (Pearson *et al.*, 1958; Steele-Bodger & Wright, 1959) with regard to the efficacy of autogenous vaccines since in Trial 1 the controls showed no change. The material was collected from all affected animals and pooled to make the vaccine, but the removal of tissue from the control animals

* M.S.E. "Atomix." Probably a domestic homogeniser would be equally efficient.

(*Concluded at foot of page opposite*)

Current Literature

BOOK REVIEWS

The Kybernetics of Natural Systems. A study in patterns of control. D. & K. Stanley-Jones. Pergamon Press, Oxford (1960). 42s.

Cybernetical concepts and methods, originally devised for the application in communication-engineering and automatic processes of control are found to be increasingly useful in physiology, medicine and psychology. Unfortunately, much of the relevant literature is made difficult to understand with complicated mathematics. In this book the laudable attempt is made to apply cybernetical concepts to biological problems in a non-mathematical way. A few simple definitions are given of negative and positive feed-back, of stability, oscillation and run-away, and these concepts are then employed to elucidate the principles of control of a number of physiological systems underlying the behaviour of invertebrate and vertebrate animals, including man.

Readers of this volume should find much of interest in the sections dealing with the control of voluntary and involuntary muscles, the working of the central and autonomous nervous systems, tremors, and the role muscle spindles and otolith organs in the maintenance of posture and in locomotion. The discussion of the problem of the primary source of neural impulses is particularly clear, though this clarity is partly achieved by the omission of data supporting the idea of spontaneous discharge of nerve cells.

The book is well written in a fluent if at times unduly repetitive style. It shows what insight can be gained with cybernetical methods even at this elementary level. In some places a diagram of the control circuits envisaged would have been helpful.

Many of the authors' cybernetical theories on human disorders (ocular diseases such as miner's nystagmus and oculo-gyric crises, tremor, poliomyelitis, etc.) are extremely interesting. While the reviewer feels not competent to judge the evidence, it would appear that the mere awareness of the possible existence of control circuits of the kind described might well be an asset in the diagnosis and treatment of these disorders.

It is a pity that some of the best analysed control systems, *e.g.* those underlying the localisation of prey by the praying mantis, the maintenance of the upright posture in fish, optomotor reactions of insects, constancy of size perception in man, etc., have not been mentioned. In these studies the mathematical treatment of the data demonstrate even more clearly the possibilities of cybernetical methods, *e.g.* in the prediction of the existence of otherwise unsuspected control mechanisms. In addition, a rigorous mathematical treatment prevents one from applying cybernetical principles too sweepingly, a tendency from which the authors are not altogether free.

On the whole the book is well suited to acquaint the physiologist and psychologist with the usefulness of the cybernetical approach. It may well encourage

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Treatment of Bovine Cutaneous Papillomata with Autogenous Vaccine.—*Concluded.*

during Trial 1 appeared to have no effect on the warts. The effect seen in Trial 2 might perhaps have been coincidental since the effect of removing warts appears to be delayed (Olson & Skidmore, 1959), but it is felt that most of the results in Trial 1 are attributable to vaccination.

Pearson *et al.* (1958) suggest 2 doses of vaccine at 14-day intervals. In these trials one dose appeared to be adequate, although results might have been more convincing had more tissue been available to give a higher concentration of virus. Olson, Segre and Skidmore (1960) found that a single dose of 5 ml. of a 5 per cent. tissue suspension in 0.5 per cent. formol-saline gave a good immunity using a single dose. Estimation of the tissue concentration used in the present work was not carried out.

Storage of the material seems to present no problems since after 3 weeks at room temperature the vaccine was still viable. Indeed, Olson *et al.* (1960) found the virus still present after 4 years in 50 per cent. glycerol-saline.

The fact that many animals lose their infection in the spring when environment and nutrition change and improve, certainly had little or no effect on these trials: the indoor group were left indoors until the end of the trials and their results were very similar to those of the outdoor group.

The length of incubation was at least 4 months. These observations agree with those of Bagdonas and Olson (1953) in that (a) the warts appeared on all the animals apparently simultaneously at the end of February, while (b) the animals of group A had had no direct contact with those of Group B during the interval from November until after the trials were completed. The incubation period could have been shorter if one could show that a large number of vectors, capable of transmitting the agent, were present in that period. This condition is only fulfilled in the very early part of the period. The other alternative of the transmission by fomites from one group to the other, in sufficient quantity to infect all animals simultaneously, seems very unlikely.

Acknowledgments.—I should like to express my thanks to Dr. R. N. Smith, Department of Veterinary Anatomy, University of Bristol and to Mr. K. Hibbitt, of the Department of Biochemistry, for their helpful criticism of the manuscript.

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people to consider these principles in the more mathematical texts, some of which are cited in the book.

Principles for British Agricultural Policy. Edited by H. T. Williams, Oxford University Press, London (1960). 18s.

At the end of the war the Nuffield Foundation brought together a group of experts to formulate a body of principles on which could be designed a long-term policy for the use of the land in the United Kingdom. It was intended to study farming policy by bringing together experts on agriculture, nutrition military strategy as it affected agriculture, and then to elaborate a future policy. The report of the expert group was delayed because many of the members of the Committee were also engaged in other duties, and in 1956 Mr. H. T. Williams, Deputy Principal and Bursar of the Seale Hayne Agricultural College was given the task of preparing a report for publication. Because of this delay, many of the recommendations in the book, and their application to present-day conditions, have already been anticipated by the expansion of the National Agricultural Advisory Services, and the very wide use now made of scientific information.

Much information has been amassed in this book, and it serves as a fascinating study of the development of agriculture in the United Kingdom since 1840. It is, therefore, a valuable source of information to students of agriculture who wish to have a picture of this industry in the pre-war period. Farming systems are discussed and the pattern is traced of agriculture in a community which has largely become industrialised.

In the discussion, special emphasis was placed on an investigation into the value of home grown food as compared with imported foods. The committee came to the conclusion that the only advantage to be obtained from the use of home grown as distinct from imported foods was that they were fresher and therefore tasted better.

Emphasis is placed on the importance of preventive methods in the control of animal disease, and these are discussed in association with grassland management and improved housing for animals. It is apposite in view of the present problems associated with fowl pest, that particular emphasis is placed on the control of diseases which can be imported from overseas. Newcastle disease is cited as an example of a disease which is now endemic in this country as a result of its import in diseased table poultry in 1947.

Because this book contains so much detail, and covers so many aspects of agricultural development, it can be thoroughly recommended to all readers interested in the history and future of British agriculture.

An Introduction to Animal Husbandry in the Tropics. G. Williamson and W. J. A. Payne. Longmans, Green & Co. Ltd., Harlow, Essex (1960). 48s.

Animal Husbandry refers to the economy and careful management of domestic animals and this book is concerned with this subject under tropical conditions, based, of course, on long experience gained in temperate climates mainly in Europe.

Basic principles are set out in the first part of the book and naturally the effect of climate on livestock is first considered. This is followed by the fundamental subjects of health, nutrition and breeding.

Part 2 is concerned with the husbandry of different types of cattle, buffalo, sheep, goats, pigs, camels and poultry which are indigenous to tropical countries, and how they differ in their requirements, under their particular environment, from European stock.

The adaptation of the well tried methods of husbandry practised for generations in Europe to the particular condition of climate and vegetation is well set out and documented. The book is eminently practical in outlook, and Part 3 is concerned with animal products as the logical sequence to the basic principles of husbandry. The account given of the various methods of production of milk, meat, hides, etc., in different countries is of considerable interest.

Because large numbers of livestock in the tropics are owned by nomadic tribesmen, and the sociological significance of cattle is of great importance to these people, there is a special chapter dealing with the nomad in West and East Africa. This is a mixture of anthropology and animal husbandry which are inseparable in this context. Marking of native stock for identification is also described.

This book has attempted, with considerable success, to solve the formidable problem of presenting in one volume the knowledge acquired over many years of livestock husbandry in the tropical zones.

The authors present the specialised knowledge of an experienced veterinarian and a scientific agriculturist with much tropical experience, together with contributions from other experts. The result is a most useful book of reference which will serve as a basis for anyone who may be called upon to take charge of or advise on the care of domestic animals in tropical climates.

The book is attractively set out and the printing and illustrations are good. The references to the literature at the end of each chapter will be found most useful and will facilitate further study of any particular aspect of the subject.

Principles and Methods of Animal Breeding. R. B. Kelley, O.B.E. Published by Angus and Robertson, London (1960). 42s.

Students and those actively engaged in the art of animal breeding will welcome this revised edition of Dr. Kelley's comprehensive book.

His efforts to maintain simplicity have been achieved by the clarity of his style, particularly in those chapters dealing with the theoretical aspects of animal breeding. The explanation of complex interactions is well illustrated by reference to examples which are met in the field and therefore of deep interest to breeders.

The author draws on a wealth of balanced knowledge which is evident in the wide coverage of this book. It may, however, surprise readers that contemporary comparison is not discussed in the sections on progeny testing.

News and Comment

RABIES CONTROL

One dog was recently vaccinated against rabies. The publicity given to this event and the consternation aroused emphasises the vigour with which the authorities enforce our rabies control legislation. Everybody knows about the 6 months quarantine requirement for imported dogs and cats. Many are unaware that the importation or use of rabies vaccine is forbidden. There has been pressure to recognise vaccination as a substitute for quarantine; nobody can doubt now that vaccination is not regarded as a practical alternative.

There have never been more animals in quarantine than at present. The total—about 1,500—is close to the capacity of existing quarantine kennels. Veterinary surgeons putting up new kennels might bear in mind this boom in imported dogs and cats. It could be advantageous to build new kennels according to the specifications demanded for quarantine purposes. They could then be used in a dual purpose capacity. Animal Health Division at Tolworth can give details of the requirements.

WARBLE FLY CONFERENCE

New methods of warble control and their practical application were discussed at a conference organised by Dow Agrochemicals at Agricultural House, Knightsbridge, London, on October 28th.

The first speaker, Mr. W. E. Bowden, of The Fatstock Marketing Corporation, emphasised the increasing importance of producing warble-free hides at a time when the leather industry faced intense competition from synthetic products. He regretted the lack of authentic figures of damage caused by warbles in Britain but estimated the loss to The Fatstock Marketing Corporation to be at least £50,000 each year.

Mr. W. G. Harvey of The Hides & Allied Trades Improvement Society explained that although the tanner off-set his loss on warbled hides by adjustments to the price paid for them, it was becoming increasingly difficult to process them economically due to the rising cost of labour. In the past, warbled hides had been used to make sole leather, but this outlet for damaged hides now represented only a fraction of the pre-war sales.

The results of 2 years' extensive field trials with the organo-phosphorus compound "Etrolene" were described by Mr. F. G. Brown, M.A., M.R.C.V.S., veterinary adviser to Dow Agrochemicals. In trials involving 800 head of cattle a single oral dose of Etrolene reduced the numbers of warbles present the following spring by 89 per cent. Of the treated animals 68 per cent. were free of warbles whilst the remainder had an average of 1 warble per beast. In contrast 77 per cent. of untreated controls were affected by warbles with an average of 8 warbles per animal. In most of the trials treated cattle gained more weight than the untreated animals. These gains were found much more regularly in beef type animals which were well fed and wintered indoors: the results were much more variable in less well fed store animals and dairy followers. The treatment reduced the number of stomach and intestinal worm eggs in the faeces and it was not certain whether the increased weight gain was due to the drug's anthelmintic action or to its effect against warbles.

Dr. W. E. Ripper said that the improved weight gains following treatment should provide sufficient inducement to owners of fattening cattle, but believed that some further incentive was necessary to encourage farmers to treat less well-nourished types of stock. He suggested that an increased subsidy should be paid at sale time to farmers who could produce a veterinary surgeon's certificate to show that their cattle had been treated systemically against warbles.

Although held during the course of the London Dairy Show, the small proportion of farmers among those present at the conference suggested a lack of interest in warble control by the majority of stock owners.

MONOFILAMENT NYLON

Monofilament nylon is now being manufactured in colours and sizes which differ from those previously obtainable. Table I shows the old scale of sizes, colours and diameters and Table II the new range of this product. It will be seen that the new 5/0, 4/0 and 3/0 are identical to the old sizes 1, 2 and 3. The thicker sizes are different in colour, diameter and code.

TABLE I
MONOFILAMENT NYLON

Size	Colour	Diameter Range
1	Black	0.004—0.006 in.
2	Blue	0.006—0.008 ,,
3	Purple	0.008—0.010 ,,
4	Green	0.010—0.012 ,,
5	Black	0.012—0.014 ,,
6	Blue	0.014—0.016 ,,
7	Purple	0.016—0.018 ,,
8	Green	0.018—0.020 ,,

TABLE II

Code	Colour	Diameter Range
5/0	Black	.004—.006 in. .10—.15 mm.
4/0	Blue	.006—.008 in. .15—.20 mm.
3/0	Purple	.008—.010 in. .20—.25 mm.
2/0	Black	.010—.013 in. .25—.325 mm.
0	Blue	.013—.016 in. .325—.40 mm.
1	Purple	.016—.019 in. .40—.475 mm.
2	Black	.019—.022 in. .475—.55 mm.

B.V.A. CAR BADGES

With reference to previous notes regarding car badges, members are advised that although these are now in production, supplies are being received in very small quantities. It is expected to take some

time to despatch badges to all members who have ordered them, and the manufacturing delay, which is outside the Association's control is regretted.

A.A. BOXES

We are grateful to those Divisions and individual members who have sent us details of incorrect or insufficient information regarding neighbouring veterinary practices, as shown in local A.A. boxes.

May we ask those who have not yet been in touch with B.V.A. Headquarters on this subject to let us know, *if possible before next Wednesday, November 16th*, of any such omissions or inexactitudes in their local A.A. boxes, with precise details as to the site and serial number of the box or boxes in question.

THE RESEARCH DEFENCE SOCIETY

The Twenty-ninth Stephen Paget Memorial Lecture entitled "Testing Drugs on Man" will be delivered by Professor J. H. Burn, M.D., F.R.S., on Tuesday, December 6th, 1960, at 5.30 p.m., in the Physiology Lecture Theatre, University College, London, Gower Street, W.C.1. All members of the Society and their friends will be welcome.

The Annual General Meeting of the Society will be held after the lecture.

THE REGISTER OF VETERINARY SURGEONS

Mr. Gordon Robert Carter, D.V.M. (Toronto), M.S. (Iowa)., c/o F.A.O. Regional Office, Maliwan Mansion, Phra Atit Road, Bangkok, Thailand, was registered in the Commonwealth List of the Register of Veterinary Surgeons on November 1st, 1960.

THE ROYAL COLLEGE OF VETERINARY SURGEONS

The Old Building

The building and historical contents of 9 and 10 Red Lion Square can be seen by anyone interested, preferably by making a previous appointment, at any time between 9.30 a.m. and 5 p.m. on Monday to Friday inclusive, up to the evening of Wednesday, November 30th.

Special arrangements by previous appointment can also be made for the building to be seen at any time on a Saturday or Sunday until that date.

PERSONAL

Mr. D. G. H. Jones, Veterinary Officer, who was stationed at Cambridge, resigned from the Ministry's service as from October 31st, 1960.

Mr. T. H. Jones, M.R.C.V.S., of the Gloucester Centre of the Milk Marketing Board, has been appointed Deputy Regional Veterinary Officer to Mr. J. P. Frappell, M.R.C.V.S., who is the Regional Veterinary Officer for the South Western Region.

Dr. D. E. Stevenson has recently resigned from the staff of the School of Veterinary Medicine, Cambridge, in order to join the Tunstall Laboratory of Shell Research Ltd. as head of the Division of Toxicology.

Births

JONES.—On October 17th, 1960, to Aileen (*née* Graham) wife of Elfed J. Jones, B.SC., M.R.C.V.S., of Tenbury Wells, Worcester, a daughter, Susan Elizabeth, sister to Richard.

WADLAND.—On November 2nd, 1960, to Jean (*née* Ash) wife of Harold Wadland, Wardington, Banbury, a son, James Richard.

WILSON.—On October 28th, 1960, at Craigtoun Maternity Hospital, St. Andrews, to Mary (*née* Mitchell) wife of H. Cowan Wilson, B.V.M.S., M.R.C.V.S., 134 Bonnygate, Cupar, Fife, a son, Harry Cowan.

COMING EVENTS

November

16th (Wed.). WARWICKSHIRE VETERINARY CLUB. Meeting in the Regent Hotel, Leamington Spa, 8 p.m.

17th (Thurs.). DUMFRIES AND GALLOWAY V.V.B.F. LADIES GUILD. Annual Buffet Dance at the Cairndale Hotel, Dumfries, 8 p.m.

V.V.B.F. COUNTY OF AYR LADIES' GUILD. Annual Buffet Dance in Western House, Ayr.

HERTS. AND BEDS. VETERINARY SOCIETY. Meeting at the George Hotel, Luton, 7.30 p.m.

19th (Sat.). B.S.A.V.A. SOUTHERN COUNTIES REGION. Meeting at the Royal Hotel, Winchester, 7.30 p.m.

23rd (Wed.). WESTERN COUNTIES VETERINARY ASSOCIATION. Annual Dinner Dance at the Imperial Hotel, Exeter, 7 p.m.

23rd and 24th (Wed. and Thurs.). B.V.A. CONFERENCE ON HYPOMAGNESAEMIA. Meetings at the Victoria Halls, Southampton Row, London, W.C.1, commencing 10.30 a.m. each day.

LAKELAND VETERINARY ASSOCIATION. General Meeting at the Fleece Inn, Kendal, 6 p.m.

24th (Thurs.). ROYAL (DICK) VETERINARY COLLEGE RUGBY CLUB. Annual Ball in the Adam Rooms, George Street, Edinburgh, 9 p.m.

25th (Fri.). WESTERN COUNTIES VETERINARY ASSOCIATION. Dinner-Dance at the Rougemont Hotel, Exeter, 7 p.m.

30th (Wed.). A.V.T. & R.W. SCOTTISH REGIONAL GROUP. Meeting at the Royal (Dick) School of Veterinary Studies, 2.30 p.m.

December

1st (Thurs.). LAKELAND VETERINARY ASSOCIATION. Annual Dinner Dance at the Old England Hotel, Bowness-on-Windermere, 7.30 p.m.

2nd (Fri.). NORTH OF ENGLAND VETERINARY MEDICAL ASSOCIATION. Annual Dinner-Dance at the White Swan Hotel, Alnwick.

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN
Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-Mouth	Fowl Pest	Sheep Scab	Swine Fever
1st to 15th October, 1960		2	—	7	45	—	41
Corresponding period in	1959	15	1	—	70	—	58
	1958	13	—	1	47	—	36
	1957	13	1	30	48	—	37
1st January to 15th October, 1960 ...		161	1	28	1,496	—	929
Corresponding period in	1959	196	2	9	610	—	983
	1958	132	5	115	584	—	1,026
	1957	270	7	94	668	—	764

TUBERCULOSIS (ATTESTED HERDS) SCHEMES

The number of Attested Herds, i.e., herds officially certified as free from Tuberculosis as at 30th September, 1960, was as follows:

ENGLAND	WALES	SCOTLAND	TOTAL (GREAT BRITAIN)
162,389	37,867	43,677	243,933

TUBERCULOSIS ORDER OF 1938

The number of bovine animals slaughtered under the Tuberculosis Order of 1938 during the period 1st July to 30th September, 1960, was 8.

B.V.A. COMMITTEE AND COUNCIL MEETINGS

Tuesday, November 15th, at 7, Mansfield Street
5 p.m. Finance Sub-Committee.

Wednesday, November 16th, at 7, Mansfield Street
10.30 a.m. Small Animals Committee.
12 noon Parliamentary and Public Relations Committee.
2.15 p.m. Veterinary State Medicine Committee.
4 p.m. Home Appointments Committee.

Thursday, November 17th, at 7, Mansfield Street
10.30 a.m. Farm Livestock Committee.
2.15 p.m. General Purposes and Finance Committee.

Friday, November 18th
10.15 a.m. Council Meeting in the Connaught Rooms, Great Queen Street, London, W.C.2.

Technical Development Committee
The next meeting of the Technical Development Committee will be held at 7, Mansfield Street, London, W.1, at 10.30 a.m. on Monday, December 5th, 1960.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak

Anthrax

Lancs. Lostock Hall Farm, Lostock, Bolton (Nov. 4).

Foot-and-Mouth Disease

Norfolk. White House, Flordon, Norwich; Martineau Lane, Norwich; Lodge Farm, Langmere, Dickleborough, Diss; Rose Cottage Farm, Tibenham, Norwich; Deal Farm, Bressingham, Diss; Four Winds, Metton Road, Felbrigg, Cromer (Nov. 7).

Northumberland. West Road, Tantobie, Newcastle upon Tyne; Eland Hall, North Farm, Ponteland, Newcastle upon Tyne; North Grange Slaughterhouse, Ponteland, Newcastle upon Tyne (Nov. 7).

Suffolk. Kiln Farm, Great Ashfield, Bury St. Edmunds (Nov. 6); Bench Barn, Clare, Sudbury (Nov. 7).

Yorks. Lund Warren Farm, Middleton-on-the-Wolds, Driffield (Nov. 7).

Fowl Pest

Bucks. Furze Wood House, Long Bottom Lane, Beaconsfield (Nov. 6).

Cambs. 34 Westfield Road, Manea, March (Nov. 1); 29 Westfield Road, Manea, March (Nov. 3); 37 Westfield Road, Manea, March (Nov. 7).

Essex. Hall Lane, Upminster Common, Upminster (Nov. 4); Maypole Croft, York Road, Horndon-on-the-Hill, Stanford-le-Hope (Nov. 6).

Glam. Penlasgarn, St. Mellons, Cardiff (Nov. 2).

Herts. Herts & Bucks Mink Farm, Upper Icknield Way, Tring (Nov. 1); Drayton Beauchamp Poultry Farm, Upper Icknield Way Tring (Nov. 4).

Norfolk. West Home Farm, Fressingfield, Diss; West End Farm, Tibenham, Norwich; Hall Farm, Banham, Norwich; White House, Weybread, Diss; Mill Lane Farm, Weybread, Diss; Grange Farm, Snetherton, Norwich (Nov. 1); The Fruit Farm, Wicklewood, Wymondham, Norfolk; Beechwood, Gt. Hockham, Thetford (Nov. 2); Elm Tree Farm, Fressingfield, Diss; Boundary Farm, Pulham St. Mary, Diss; Chestnuts Farm, Denham, Diss (Nov. 3); Park Farm, Winfarthing, Diss; No. 3 Site Hillcrest, Wicklewood, Wymondham (Nov. 4); The Elms, Starston, Harleston; Foxbow, Northall Green, Dereham; Martins Farm, Wingfield, Diss (Nov. 5); Springfield House, Westfield, East Dereham; Near Chapel, High Street, Wicklewood, Wymondham; Waterloo Cottage, Long Lane, Tibenham, Norwich; Church Farm, Carlton Rode, Norwich; 14 Royden Fen, Diss (Nov. 6); Blacksmiths Shop, Kimberley, Wymondham; Laurel Farm, Weybread, Diss; Low Street Farm, Hardingham, Norwich; Corner Farm, Weybread, Diss; Holly Farm, Mattishall Road, Dereham; Hingham Road, Great Ellingham, Attleborough; Knights Farm, Fressingfield, Diss (Nov. 7).

Salop. Prees Green Post Office, Whitchurch; Booley Lodge, Stanton, Shrewsbury (Nov. 4); Booley Farm, Stanton, Shrewsbury (Nov. 5); Brickyard Farm, Shawbury, Shrewsbury (Nov. 6).

Somerset. The Holly Bush, Leigh-on-Mendip, Bath; St. Donats, West Pennard, Glastonbury (Nov. 2); South Somerset Poultry Farm, Martock (Nov. 6).

Staffs. Blakeley Lane Farm, Dilhorne, Stoke-on-Trent (Nov. 4); Greenhead Farm, Kingsley Moor, Cheadle, Stoke-on-Trent (Nov. 5).

Warwicks. Wharley Hall, Barston, Solihull (Nov. 5).

Swine Fever

Berks. Common Farm, Wyfold, Checkendon, Reading (Nov. 2).

Ches. Standrick Hill Farm, Higher Heyrod, Stalybridge (Nov. 1); Lower House Farm, Mottram St. Andrew, Macclesfield (Nov. 2); Hazell Knoll Farm, Torkington Road, Hazell Grove, Stockport (Nov. 7).

Denbigh. Green Farm, Rossett, Wrexham (Nov. 3); Bedwell Hall, Cross Lanes, Marchwiel, Wrexham (Nov. 7).

Durham. Holywell Farm, Croxdale (Nov. 3).
Leics. 13 Brookside East Leake, Loughborough (Nov. 2).
Lincs. Rose Farm, Scartho, Grimsby (Nov. 1); Bilyards Farm, Harpswell, Gainsborough; Church Farm, Toft Newton, Market Rasen (Nov. 4); Elsham Aerodrome, Elsham, Brigg; Church Farm, Toft Newton, Market Rasen (Nov. 7).
Notts. Great North Road, Weston Newark; Scarthing Moor Mill; Weston, Newark (Nov. 1).
Somerset. The Piggeries, Mudgley Wall, Rooksbridge, Axbridge (Nov. 3).
Suffolk. Charmans Farm, Risby, Bury St. Edmunds (Nov. 2); Camps Heath, Oulton, Lowestoft (Nov. 3).
Sussex. Denne Meadow Farm, Southwater, Horsham (Nov. 2).
Warwicks. Lyebridge Farm, Alvechurch, Birmingham (Nov. 3).
Yorks. Thorpe Hall Farm, Thorpe Stapleton, Leeds; Whitwell Farm, Whitwell, Scorton, Richmond (Nov. 1); Swale Farm, Catterick, Richmond (Nov. 2); Sheepcote Farm, Darlington Road, North Allerton; Lowden House, Tunstall, Richmond; Golden Fields, Sheriff Hutton; North End Farm, Coniston, Hull (Nov. 3); Piggery Wayside, Old Earswick (Nov. 4); Lower Lane Piggeries, Bradford; Low Merrybent, Melsonby, Richmond (Nov. 7).

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

SCIENTIFIC

Sarcoptic Mange in Pigs

Sir,—In his paper in THE VETERINARY RECORD of October 15th, Mr. E. A. McPherson has drawn attention to the high incidence of infection with *Sarcoptes scabei* var. *suis* in pigs in this country.

We can confirm his findings from our own observations in this area. These began some 6 years ago when we were investigating the condition called nutritional dermatitis by Thomas and Eden (1954) and parakeratosis by Kernkamp and Ferrin (1953) and which was shown later to be due to a deficiency of zinc in the diet.

Our first reaction on seeing outbreaks of "dry-feed disease" as it was sometimes called was to eliminate mange by examining skin scrapings. We were able to buy, or were given, badly affected pigs from the earlier outbreaks and on examining these thoroughly at the laboratory we recovered mange mites in a high proportion of cases.

Scrapings taken under farm conditions might or might not be positive but if either a fresh carcase were available or a pig could be taken to the laboratory and examined thoroughly under light general anaesthesia, mange mites could often be recovered from some part of the body. Only a few might be found after extensive sampling and concentration of the material, in other cases they were plentiful, but they could be found in so many cases that they had to be ruled out as a factor in the disease. This was done by thorough treatment of pregnant sows and later of their litters with insecticide, and it was clear that the disease could occur in pigs quite free of mange.

About this time Tucker and Salmon (1955) showed that parakeratosis was due to a relative deficiency of dietary zinc, particularly in the presence of high dietary calcium, and we were able to obtain skin scrapings and samples of rations from 6 affected herds for analysis. In 3 of the 6 outbreaks mange mites were present and the dietary zinc level was low enough and the calcium high enough to be pathological (Zn 53 p.p.m., 52 p.p.m. and 48 p.p.m.; CaO 1.6 per cent., 2.05 per cent. and 2.4 per cent. respectively). In one outbreak mange mites were not found, zinc and calcium were 45 p.p.m. and 1.37 per cent. respectively indicating uncomplicated parakeratosis; in the fifth mange mites were plentiful and the zinc level was well over 200 p.p.m. and in the sixth mange mites were found fairly readily and the ration contained 60 p.p.m. Zn and 1.4 per cent. CaO which probably represents marginal zinc deficiency.

Out of a total of 20 outbreaks of dermatitis examined, most of them before the rôle of zinc in parakeratosis was known, mange mites were recovered in 17, sometimes in such small numbers as to make it unlikely that they were responsible, and in other cases they were numerous enough to justify a diagnosis of clinical mange.

If dietary mineral levels had been determined in the earlier outbreaks it is likely that more cases of zinc deficiency would have been confirmed in those herds in which mange mites were found only after extensive sampling, and we were almost certainly dealing in different herds with uncomplicated zinc deficiency, simple sarcoptic mange, zinc deficiency along with sarcoptic mange and cases in which the clinical dermatitis was due to zinc deficiency but in which small numbers of mange mites were present though causing no clinical disease.

More information is required on the economic importance of such a widespread disease. We have seen acute outbreaks in sucking pigs severe enough to cause death or permanent stunting, and less acute outbreaks accompanied by intense skin irritation, reduced appetite and diarrhoea. In other less severe cases there are varying degrees of restlessness, rubbing and general unthriftiness.

There is usually a marked response within 24 hours of treatment and this should be thorough in order to eliminate the infection. It has been our experience that BHC at the strength used for sheep scab control (0.016 γ Isomer) is too low for the control of sarcoptic mange in the pig even when used as a dip; it will produce a remission of symptoms but these usually recur in 3 to 4 weeks time. We have found that a single thorough treatment with 0.1 per cent. *gamma* BHC spray will eradicate this mite this confirms the findings of Roberts and Rogoff (1953).

We have not found it necessary to apply any separate treatment to the ears, care being taken to direct the spray at and into the ears of the pigs which are sprayed in small groups. We have not confirmed the total elimination of mites by long term observation as reported by McPherson but the treatment has not been followed by clinical breakdown.

In breeding units the disease should be regarded as a herd disease and treated on a herd basis to avoid reinfection from animals with quiescent disease, and in fattening units maintained by buying store pigs it is advisable to spray each intake with BHC, a practice which promotes thriftiness and which some believe reduces the incidence of fighting when strange litters are mixed.

Yours faithfully,
W. M. BROWNLIE,
I. R. HARRISON.

Boots Pure Drug Co. Ltd.,
Research Department,
The Priory,
Thurgarton,
Nottingham.
November 5th, 1960.

References

KERNKAMP, H. C. H., & FERRIN, E. F. (1953). *J.A.V.M.A.* **123.** 217.
ROBERTS, I. H., & ROGOFF, W. M. (1953). *Ibid.* **123.** 227.
THOMAS, G., & EDEN, A. (1954). *Nature.* **174.** 553.
TUCKER, H. F., & SALMON, W. D. (1955). *Proc. Soc. Expt. Biol. Med.* **88.** 613.

Prolonged Feeding of Calcined Magnesite

Sir,—The following report of a condition in yearling calves receiving calcined magnesite may indicate the possible danger of prolonged feeding of magnesium supplements.

The calves in question had been housed from the end of September, 1959, and fed a diet of good quality hay, indifferent quality silage and crushed oats. In addition, each animal received ⅓ to ⅔ ounces of cod liver oil and ½ oz. calcined magnesite per day.

Two months later, a number of calves developed stiffness and stilted gait, the symptoms being progressive. Two of the animals died, one on December 20th, 1959, and one on January 27th, 1960. Two further cases (Nos. 1 and 2) became recumbent and unable to rise. They were destroyed for *post-mortem* examination on February 17th and 19th respectively.

Post-mortem examination of calf No. 1 showed no obvious abnormality in either the respiratory or digestive organs. The urinary bladder was very distended and the kidney cortex very pale. There were numerous subepicardial, myocardial and subendocardial haemorrhages. A careful search was made for lesions of muscular dystrophy in both cardiac and skeletal muscle but none was found. Both shafts of the ilium and the ulna on one side were fractured. There were exostoses on several ribs and on the mesial surface of the scapula of one side. The epiphiseal cartilage of the tibia appeared slightly thicker than normal.

Calf No. 2 had a septic arthritis of one hock joint, possibly associated with prolonged recumbency. *Post-mortem* examination revealed no gross abnormalities of the internal organs or of skeletal or cardiac muscle. The femur of one side was fractured and exostoses were present on the ribs similar to those seen in calf No. 1.

There were 2 other calves (Nos. 3 and 4) in the same group which were stiff and whose backs were arched.

As demonstrated by the following data, serum Ca and blood inorganic P levels were abnormally low in all 4 calves.

Calf No.	Serum Ca mg. per 100 ml.	Blood Inorganic P mg. per 100 ml.
1	3.7	4.5
2	5.3	3.1
3	6.0	2.8
4	7.2	4.2

The liver Cu was within the normal range in the calves subjected to *post-mortem* examination, Nos. 1 and 2.

The other calves appeared normal. Three of these gave Ca and P blood levels as follows:—

Calf No.	Serum Ca mg. per 100 ml.	Blood Inorganic P mg. per 100 ml.
5	8.1	4.5
6	8.8	5.0
7	8.9	—

The advice given to the owner was to withdraw both the calcined magnesite and the cod liver oil and to feed ¼ oz. bone meal per head per day. Within a month the calves' appetites and general condition had improved. On March 18th, 1960, the 2 badly affected calves (Nos. 3 and 4) were bled with the following biochemical results:—

Calf No.	Serum Ca mg. per 100 ml.	Blood Inorganic P mg. per 100 ml.
3	5.5	3.4
4	12.5	6.1

No. 4 made a complete clinical recovery but No. 3 has remained somewhat stiff and decidedly stunted.

The lesions and the Ca and P blood levels are consistent with a diagnosis of rickets. However, cod liver oil would normally be expected to prevent this condition. Straightforward aphosphorosis also seems unlikely there being little reason to suppose that dietary P was grossly deficient, and while a complete analysis of the feed was not carried out, the hay gave a value of 0.2 per cent. P.

In an investigation into an outbreak of "milk lameness," McTaggart (1959) concluded that the effects of P deficiency in high-yielding cows were accentuated by a number of factors including magnesium feeding. He cited Palmer *et al.* who demonstrated a depletion of body Ca in dairy cattle receiving a Mg supplement when the dietary P was low, and Kramer *et al.* who showed that "even low concentrations of Mg can inhibit calcification of bone." It would seem that the feeding of calcined magnesite was an important contributory factor in the production of the condition described above.

Calcined magnesite has proved a valuable preven-

tive against hypomagnesaemic tetany in the case of dairy cows and it is recommended that this supplementation be confined to the "danger period" which in Ireland usually extends over the first 6 weeks of grazing in the spring. It is customary to begin supplementation about a week before the cows go out to pasture to accustom them to the taste of calcined magnesite. The continuous feeding of magnesium salts to ruminants outside danger periods is not to be recommended.

Yours faithfully,
N. E. DOWNEY.

The Agricultural Institute,
Animal Production Division,
Thorndale,
Beaumont Road,
Dublin.

November 1st, 1960.

Reference

McTAGGART, H. S. (1959). *Vet. Rec.* **71**. 709.

The New Zealand Lambing Instrument

Sir,—During the past year an obstetric device for ewes, sold under the trade name of "The New Zealand Lambing Instrument" has been selling on the Tasmanian market. Possibly a similar instrument is being offered in the U.K. but one has not seen any reference to it in THE VETERINARY RECORD or in British agricultural publications.

The instrument sold here consists of a plastic hollow tube 2½ inches in circumference for a foot of its length, then bifurcating into tubular 3-inch branches of smaller calibre, like the fork of a boy's catapult.

About 4 inches from the proximal closed end there is an opening into the tube into which a 3-foot length of plastic cord is passed. The cord leaves the tube at the end of one of its forks and returns by the other fork to form a terminal loop. It then goes back along the main tube to emerge by the hole in the shaft and join the other end of the plastic cord in a plastic handle.

By pulling or pushing the handle one can enlarge or diminish the end-loop as required for snaring and holding the head of the foetus within the uterus, either alone or incorporating both fore legs. The device is not an obstetrical cure-all—nothing is—but it is useful on occasions as the following typical case history will show.

A call came one evening from a neighbouring eminent breeder of Dorset Horns for help in lambing his best show Dorset ewe. He was having difficulty in getting out the head of a ram lamb and had mislaid the wood and wire contraption he depended on in such cases. The breeder explained that as there was obviously not enough room for fore legs and head to come together into the pelvis he had corded and repelled both fore legs into the uterus. The two ends of the cords were seen hanging from the lower commissure of the vulva.

Recent articles on ovine obstetrics in THE VETERINARY RECORD have shown some difference of opinion about the advisability of cording and repelling fore legs, but it is commonly and often successfully done by leading Tasmanian breeders for delivering Dorset Horn ram lambs.

It sometimes happens here, and perhaps also in the U.K., that the horn bud on a large ram Dorset Horn foetus is so large as to form an obstruction in transit from the uterus into the pelvic canal. It would appear that during parturition pressure against this obstruction causes the nose of the foetus to dip below the rim of the pelvis while the fore legs pass on to appear at, or near, the vulva.

When this happens and the lamb is still alive the experienced breeder, as in the present case, puts a slip knot (usually of bagging-twine) around the pastern of the fore leg and reinforces the grip by a half-hitch just above the hoof. Both fore legs are then repelled to the fullest possible extent and work begins on getting out the head alone if possible. Sometimes this can be done by finger and thumb eye-grip, but not always, as in the present case where both the breeder and I failed to move the lamb by this means. The vulsellum forceps was then applied and pulled to the limit of safety for the lamb, but this also failed.

It was then decided to try the New Zealand lambing forceps, always now carried in my surgical kit, but never tried except for live lambs. After 2 failures the plastic loop of the instrument was passed well behind the horn buds and the poll while the plastic fork was pushed forward under the muzzle of the lamb to keep the nose above the bony inlet to the pelvic canal when traction should begin. The plastic cord was simultaneously tightened by the right hand outside the ewe.

Presently the head began to move into the pelvis and, after some manipulation of the surrounding parts, was brought outside the vulva. Then came the anxious moment of trying to get out both fore legs by pulling on the cords attached to the feet. Traction on the first cord failed—the foot would not budge. Accordingly, this foot was abandoned and traction tried on the other cord, whereupon a foot quickly and easily emerged. The space thus enlarged, traction on the first cord soon succeeded, and presently a live and viable lamb was delivered. This wholly successful delivery of a live lamb in these circumstances could not have been done so easily and safely by any other means so far tried here.

The value of the plastic cord is that it can be felt and manipulated readily at all times within the uterus, whereas string tends to become imperceptible to touch, and incapable of easy manipulation, when wetted with natural and lubricating fluids.

The value of the plastic fork in keeping up the nose of the lamb during traction is also obvious in these all too frequent cases where the canal is too narrow, or the hand too large, to permit manipulation during transit from uterus to and through pelvis.

The value of the New Zealand Lambing Instrument, however, is limited to the saving of a live lamb in an uncomplicated case. When the lamb is dead, or there are other complications, the instrument has been found of small use and the other means are preferable. One of our largest and most famous

Tasmanian breeders told me recently that though he always had the New Zealand Instrument at disposal he had only used it 3 times in the past season.

My own experience has been similar: there is just the occasional case, as in the instance just given, where there is nothing else quite so good.

Yours faithfully,
J. W. RAINEY.

P.O. Box 67,
Westbury,
Tasmania.
October 27th, 1960.

The Racing Greyhound

Sir,—I thank Mr. Holmes for his query. There are two essential factors in this administration process which should be borne in mind. These are: (1) that all the Greyhounds in our kennels are over 15 months and the bulk are between the ages of 2 to 3 years. There are some, too old for racing, which are retained in the kennels for stud purposes. Factor (2) is that there is no distemper nowadays (9 cases have been recorded in 1958 and 1959). This leaves the field quite clear of complication. In this distemper-free period (the number above excepted) to date over 6,000 dogs have come into the kennels and a similar number have gone out. The kennel strength averages 600. Death rate has been infinitesimal.

Tonsillitis is probably the most common type of sickness affecting the dogs. It has a nuisance value for it is often accompanied by some muscular pain. Affected dogs are rarely hospitalised and recovery takes place in about 3 days.

Doubless because the majority of our dogs, being mature, have already travelled across the country and around it, blood samples of a group taken at random will show about 90 per cent. positive for agglutinins of C.V.H. There can be no point in vaccinating such animals, therefore no hepatitis vaccine is used on our premises. Supported by an excellent picture of health I ignore C.V.H. altogether and the dogs pass through their racing careers without being visibly (to me) affected. They do refuse a meal on occasion and one sees the very occasional case of haemorrhagic enteritis, but "blue eyes" and other classical symptoms of C.V.H. are unknown.

Most of the dogs in the kennels are privately owned. They are bought in Eire, at public auctions in London, or sometimes are privately reared. There is a separate group—much smaller—reared for the Company, whose health is under my control from birth. They are whelped in Surrey, immunised against distemper and leptospirosis icterohaemorrhagica then sent to Cumberland and Westmorland to be reared. I believe that one sapling only in the last few years has died of a condition diagnosed as C.V.H. by the local veterinary surgeon. None is vaccinated against C.V.H., and eventually all those which are good enough find their way into our own and other racing kennels after being re-vaccinated against distemper only. Their subsequent history follows the same pattern as the others.

I am aware, of course, that a number of present-day Greyhounds may well be vaccinated against C.V.H. by one or other of the vaccines, live or dead. But this letter deals with the period mid-1955 until to-day and therefore includes a period of time before C.V.H. vaccines were on the market.

In view of my experience as described, and it will be realised that other veterinary colleagues have seen it also, I answer Mr. Holmes by saying that economically in my conditions in a large kennel of Greyhounds, properly fed, C.V.H. plays so small a part as to be ignored and is of no importance.

It seems possible that dietetic conditions or some other factor (breed incidence?) obtaining in a Greyhound kennel such as ours, allows the dogs to shrug the disease off.

I consider it to be an interesting pathological entity capable of being dangerous at *post-mortem* in that findings of a high titre in the blood *may* be wrongly construed as having played a responsible part in the cause of death.

Yours faithfully,
JAS. K. BATEMAN.

10 Brackendale,
London, N.21.
October 31st, 1960.

Eradication of Disease

Sir,—From the paper on brucellosis eradication in Northern Ireland by W. R. Kerr (*Vet. Rec.* **72**. 921), farmers wishing to join the scheme will be required to maintain records of the animals' movements and test results.

Surely if this had been enacted by legislation years ago the present problems of disease eradication would have been lessened considerably? Furthermore, it would be interesting to know from a member of the Parliamentary Committee of the B.V.A. why such legislation cannot be made *now* for the maintenance of records for *all* veterinary treatment and movement of animals. This, surely, would ultimately produce a picture of the distribution and intensity of any disease or health problem in the country as a whole, thereby producing better co-ordination between practitioners, laboratories and administrators.

Yours faithfully,
DEREK D. POUT.

Royal Veterinary College,
Royal College Street,
London, N.W.1.
November 6th, 1960.

Laryngeal Obstruction in the Calf

Sir,—The Clinical Note in your issue of October 22nd by Seawright and Doyle prompts us to record that in the past 2 years we have had 4 similar cases in this area, all of them 6-month-old Aberdeen-Angus cross calves at grass. Calf F.C. was slaughtered 8 days after onset of symptoms, the others died: M.D. 6 days, C.T. 9 days, and M.S. 3 hours after onset. Treatment included tracheotomy in 2 cases (F.C. and M.D.) and this gave some relief and postponed death. Drugs used included mepyramine maleate, promethazine hydrochloride

potassium iodide, prednisolone, sulphadimidine, streptomycin, chloramphenicol, oxytetracycline, and adrenaline.

Post-mortem examinations were carried out on C.T. and M.S. In C.T. the tissues around the larynx were dry, crumbly and necrotic. In M.S. the tissue surrounding the larynx was a "bloody gelatinous mass." No organisms, aerobic or anaerobic were isolated.

Space does not permit detailed case histories but one comparison must be made with that described by Seawright and Doyle. In our series of cases the cause of the obstruction lay outside the larynx and was due to swelling of the peri-laryngeal tissues sometimes involving the pharynx and tongue. Could it be that the initial cause is of a traumatic and/or allergic nature (with, if the animal lives long enough, secondary infection by *Fusiformis necrophorus*) rather than the calf diphtheria syndrome suggested by Seawright and Doyle? If so, is there any significance in the common age-incidence (6 months) or in the locality, Nairn and Keith being 37 miles apart?

Yours faithfully,
J. G. WATSON,
M. MARSHALL.

Craig Royston,
Nairn.
November 2nd, 1960.

Reference

SEAWRIGHT, D., & DOYLE, J. *Vet. Rec.* **72**. 904.

A Veterinary Centre

Sir,—I have been surprised to note the lack of response to the news you gave in a recent issue concerning the sale of 10 Red Lion Square. The most satisfactory sum obtained for the freehold of these premises stands greatly to the credit of the Officers of the Royal College, and no doubt to the tenaciousness of the Registrar in refusing to accept the earlier offers, however attractive they may have seemed. Every member of the profession must regret the passing of this familiar land-mark in our professional life. However, what a splendid opportunity it provides for the men of vision in our profession to create in London a Veterinary Centre which could become the Mecca for professional colleagues from all over the world.

The suggestion I would like to make may appear to be somewhat radical, and there may be some who for "political" reasons consider it most impractical. The great advantages, however, do, in my view, far outweigh any small difficulties that may arise. I would propose Sir, that the B.V.A. join forces with the R.C.V.S. to create a joint headquarters in London for both organisations permitting the sharing of clerical and typing facilities and other overhead expenditure, the provision of a meeting place and conference hall in keeping with the great tradition of our profession, and even more important, providing for the considerable future demands for administrative facilities in these days of rapid expansion. No one can claim that 7 Mansfield Street is either dignified or even impressive; in fact, it is really no more than a *pied à terre*. I am sure that the hard-working members of the B.V.A. office staff would be the first to agree that the accommodation has considerable limitations.

The adoption of my proposal would permit the pooling of the financial resources of the R.C.V.S and B.V.A. to create a building worthy of our professsion permitting the Royal College to be housed on one floor, the R.C.V.S. Library on another, and the B.V.A. on another with additional accommodation available, perhaps, for administrative facilities for the non-territorial Divisions of the B.V.A. which appear to be growing rapidly. These Divisions will soon require the services of part-time salaried secretaries, and with suitable accommodation the different Divisions could share such a person and his office staff. The practical advantages of this suggestion in improving efficiency will be obvious. It might also be possible to have some club facilities, such as a small restaurant and bedrooms for members' use when attending meetings in London.

As a parallel, I would refer you to the organisation which existed, until recently, for the administration of the British Turkey Federation and the British Broiler Growers Association. Both these organisations were run by a joint secretariat administered by a limited company known as Poultry Administration Limited. Could not such a system be adopted for our purposes? A limited company could be formed with the B.V.A. and R.C.V.S. as major shareholders, and if the capital available was inadequate, shares in the company could be made available to members of the profession. This company could then run the Veterinary Centre receiving revenue by way of rents and sub-letting of accommodation, meeting rooms, etc., the intention being to cover costs rather than to show a substantial profit. I realise that the financial difficulties involved in relation to income tax, the Charities Act, etc. would have to be closely investigated, but if such a scheme would produce a really first class Veterinary Centre for our profession, I am sure it would be well worth while.

I am certain, with the close co-operation that we are constantly assured exists between the B.V.A. and the R.C.V.S the planning and organisation required for the adoption of this suggestion would be carried out with the maximum of efficiency between both these bodies.

Our profession is growing fast, its social and scientific status increases daily—nothing less than a Centre such as I have described will satisfy our needs in the years to come. We have been presented with a unique opportunity which may never be repeated. Let us hope that we shall not fail through temerity or apathy to seize it eagerly and thereby create a permanent symbol of our faith in the future. The founders of our profession were never short of courage or vision; may we prove ourselves worthy of their trust.

G. N. HENDERSON.

"Birkwray,"
Oak Grange Road,
West Clandon,
Surrey.
November 2nd, 1960.